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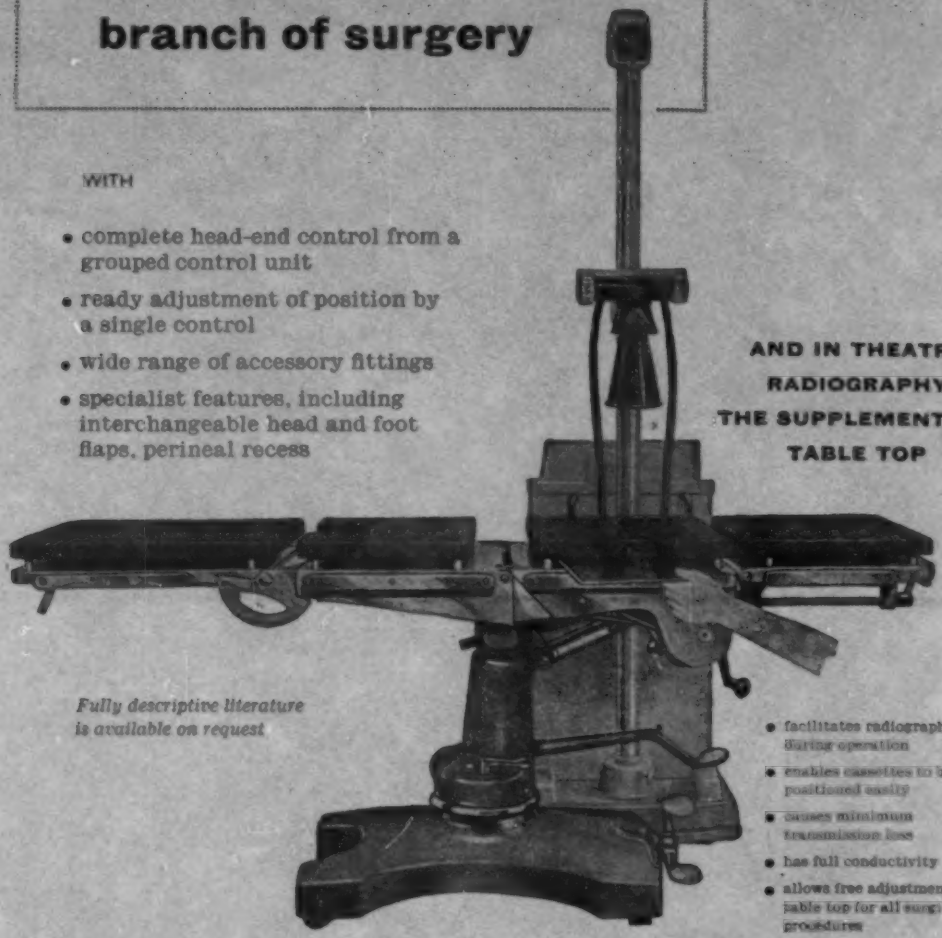

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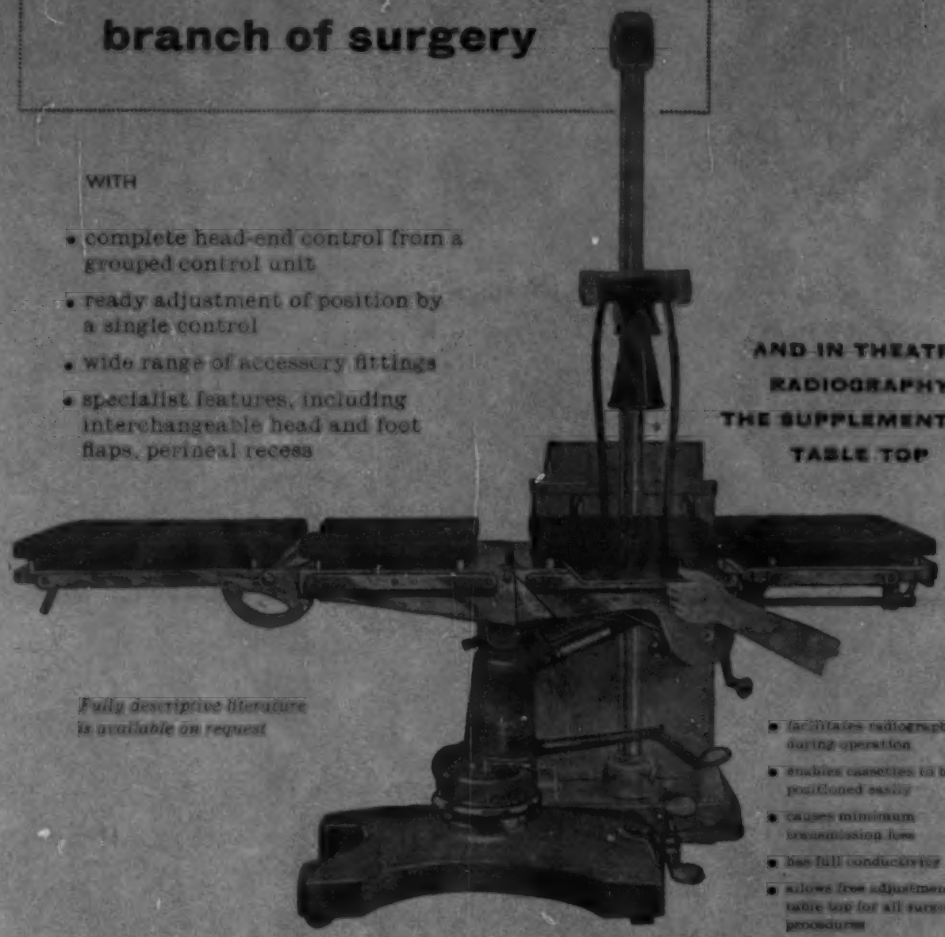

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CONTENTS, MARCH, 1960

ORIGINAL PAPERS

	PAGE
THE SURGERY OF THE ŒSOPHAGUS - - - - - - - - *Sir Heneage Ogilvie*	457
THE INNERVATION OF THE CARDIA AND LOWER ŒSOPHAGUS IN MAN *F. G. Ellis, R. Kauntze, and J. R. Trounce*	466
EXCISION OF THE MAJOR DUCT SYSTEM FOR BENIGN DISEASE OF THE BREAST - - - *John Hadfield*	472
DUPLICATIONS OF THE ALIMENTARY TRACT - - - *R. Basu, Isabella Forshall, and P. P. Rickman*	477
DIVERTICULAR DISEASE OF THE COLON - - - - *D. B. Brown and W. F. Toomey*	485
SOLITARY DIVERTICULUM OF THE CÆCUM AND ITS COMPLICATIONS - *D. B. Brown and W. F. Toomey*	493
RUPTURE OF PANCREATIC CYSTS - - - - - - - *William H. Hanna*	495
A VESICAL CALCULUS CONTAINING METHYLENE BLUE - - - - - *G. A. K. Missen*	499
NEW CONCEPTS ON AMŒBIC ABSCESS OF THE LIVER - - - - - *Milroy Paul*	502
HIATUS HERNIA: A CAUSE OF GASTRO-INTESTINAL HÆMORRHAGE - - - *Paul Marchand*	515
MESOCOLIC FIBROSARCOMA - - - - - - - - *J. I. Pugh*	526
SPONTANEOUS DISAPPEARANCE OF LUNG METASTASES IN A CASE OF KIDNEY CARCINOMA (HYPERNEPHROMA) *M. F. Nicholls and A. H. M. Siddons*	531
THE USE AND RISKS OF THE ROUX-EN-Y ANASTOMOSIS IN THE RELIEF OF POST-GASTRECTOMY SYMPTOMS *J. H. Conyers, R. A. Hall, D. Laing, C. N. Pulvertaft, and J. K. Willson-Pepper*	533
LIPOSARCOMA OF THE GREATER OMENTUM - - - - - - - *W. A. T. Robb*	537
RENAL TUBERCULOSIS - - - - - - *R. I. Harris, W. K. Kerr, and H. S. Coulthard*	539
PRIMARY FIBROSARCOMA OF THE TONGUE ASSOCIATED WITH SYPHILITIC LEUCOPLAKIA - - *R. A. Cawson*	543
SUBMUCOSAL VASCULAR PATTERNS OF THE ALIMENTARY TRACT AND THEIR SIGNIFICANCE *P. S. Boulter and A. G. Parks*	546

EXPERIMENTAL SURGERY

THE ABSORPTION OF SURGICAL CATGUT - - *Peter Lawrie, G. Elspeth Angus, and A. J. M. Reese*	551
WOUND SHAPE AND TISSUE TENSION IN HEALING - - - - - *George T. Watts*	555
CHANGES IN GASTRIC ACIDITY AND EPITHELIAL REGENERATION FOLLOWING EXCISION OF THE GASTRIC MUCOSA IN THE DOG - - - - - - *G. W. Milton, G. A. Maxwell, and E. S. Finckh*	562
HÆMOSTATIC PATCH IN PARTIAL NEPHRECTOMY IN DOGS - - - - *Norman W. Struthers*	567

SHORT NOTES OF RARE OR OBSCURE CASES

TRAUMATIC CHYLOTHORAX - - - - - - - *G. B. Ong and T. C. Lee*	570
A CASE OF PIGMENTED CONGENITAL EPULIS - - - - *T. Cradock Henry and Martin Bodian*	574
HÆMANGIOMA OF THE BLADDER - - - - - - - *J. P. Partridge*	577
POLYPOSIS OF THE SMALL INTESTINE, WITH PIGMENTATION OF THE FACE, BUCCAL MUCOSA, AND DIGITS *J. Oldfield*	578

REVIEWS AND NOTICES OF BOOKS

L'Arteriopatia Ostruttiva Aorto-Iliaca—*Ennio Muntoni, J. Del Moro, R. Liscia, and F. Pizzetti.* Osteochondritis Dissecans—*I. S. Smillie.* The Year Book of General Surgery 1959–1960—ed. *Michael E. de Bakey.* The Surgical Treatment of Scoliosis—*Louis A. Goldstein.* Die Wirbelsaulenleiden und ihre Differentialdiagnose—*J. E. W. Brocher.* Le Nouveau-Né—*Marcel Fèvre.* Les Entretiens de Bichat 1959. Outline of Orthopædics—*John Crawford Adams.* Instructional Course Lectures (Vol. XVI. 1959)—ed. *Fred C. Reynolds.* The Foot and Ankle—*Philip Lewin.* Cancer (Vol. VI)—ed. *Ronald W. Raven.* The Treatment of Bronchial Neoplasms—*Robert R. Shaw, Donald L. Paulson, and John Lester Kee.* Pediatric Neurosurgery—ed. *Ira J. Jackson and Raymond K. Thompson.* Anatomy, Regional and Applied—*R. J. Last.* Cancer of the Breast—ed. *Willard H. Parsons.* The Royal Edinburgh Hospital for Sick Children 1860–1960—*Douglas Guthrie.* Metals and Engineering in Bone and Joint Surgery—*Charles Orville Bechtol, Albert Barnett Ferguson, and Patrick Gowans Laing.* Thérapeutique Chirurgicale des Ictères par Rétention—*P. Goinard and G. Pélissier.* The Surgical Treatment of Facial Injuries—*Varaztad Hovhannes Kazanjian and John Marquis Converse.* Chirurgie der Verengungen der Oberen Luftwege—*Aurel Rethi.* Surgery of Repair as applied to Hand Injuries—*B. K. Rank and A. R. Wakefield.* Metabolic Homeostasis—*Natham B. Talbot, Robert H. Richie, John D. Crawford, and Edith S. Tagrin.* Atlas Aide-Mémoire d'Anatomie—*H. Rouvière.* Management of the Aged Surgical Patient—*Sidney E. Ziffren.* Fundamental Techniques of Plastic Surgery and their Surgical Applications—*Ian M. McGregor.* - - - 579

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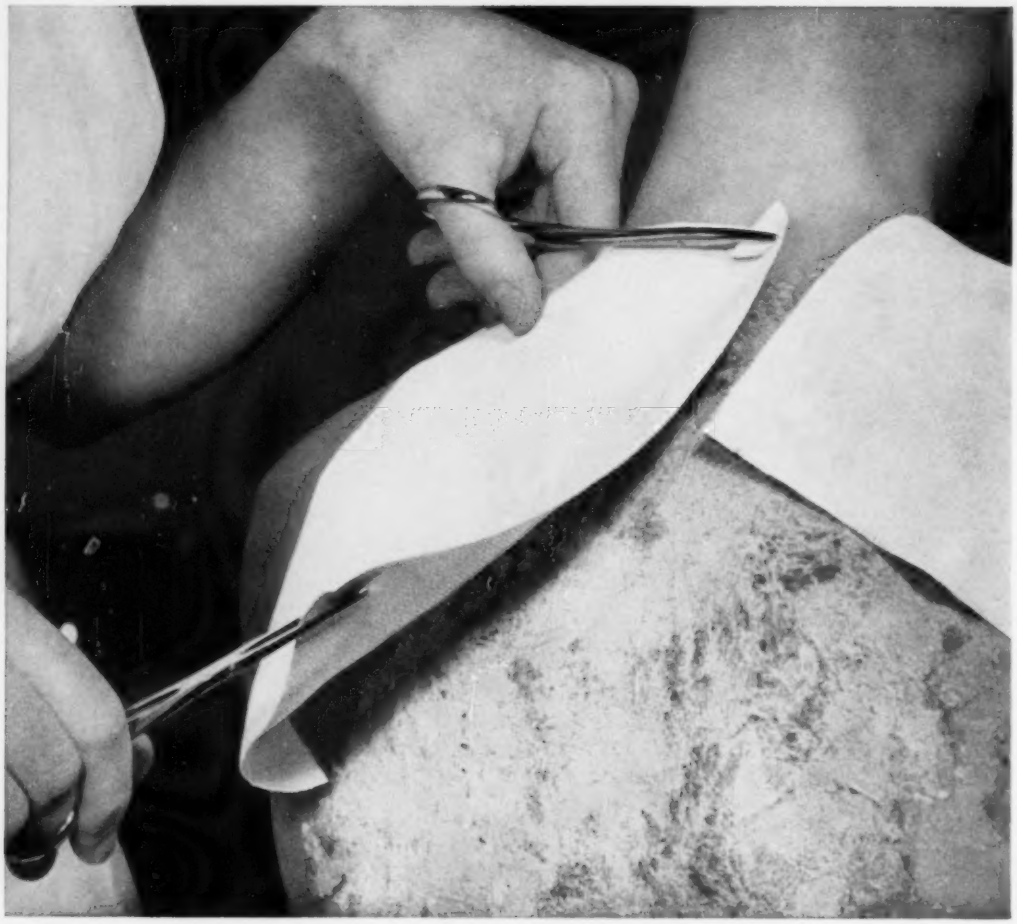

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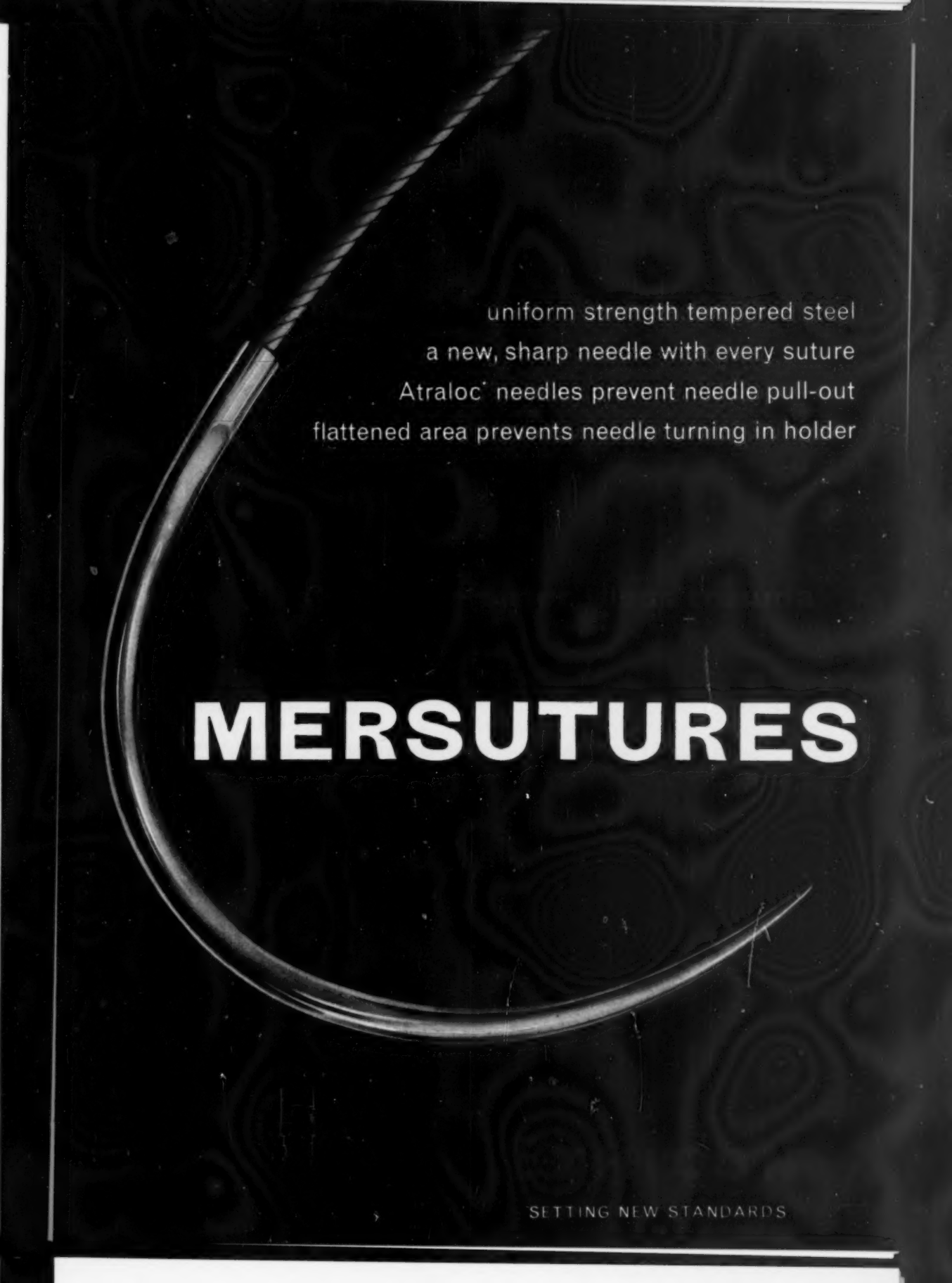
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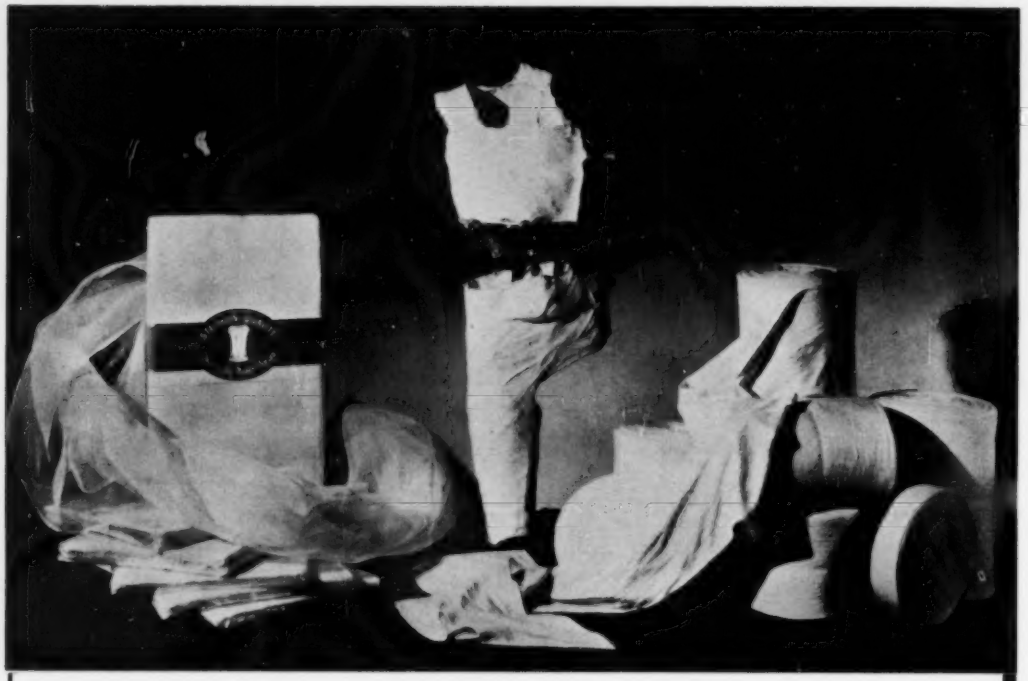

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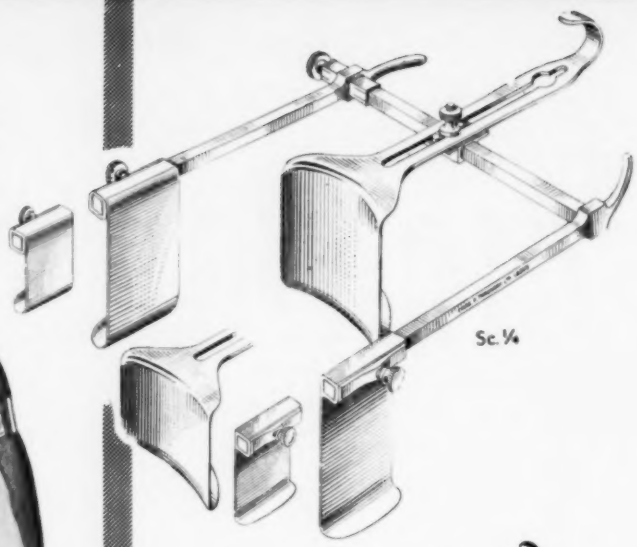

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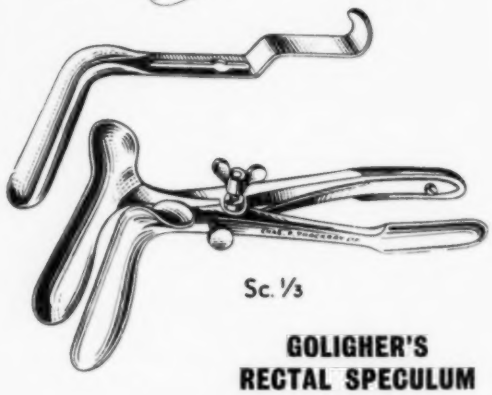

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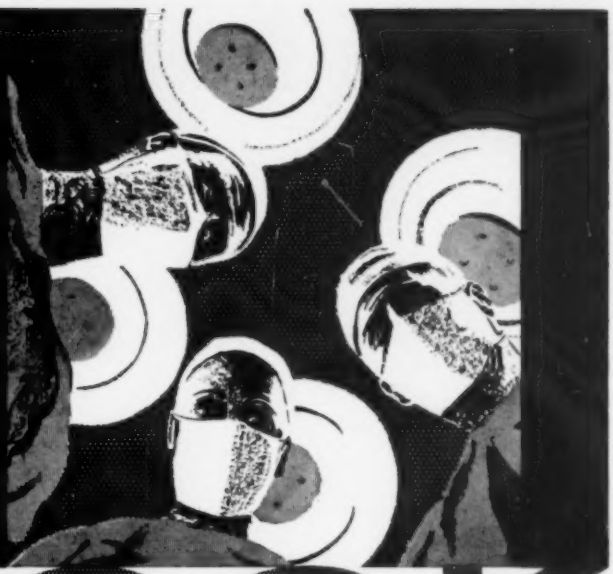

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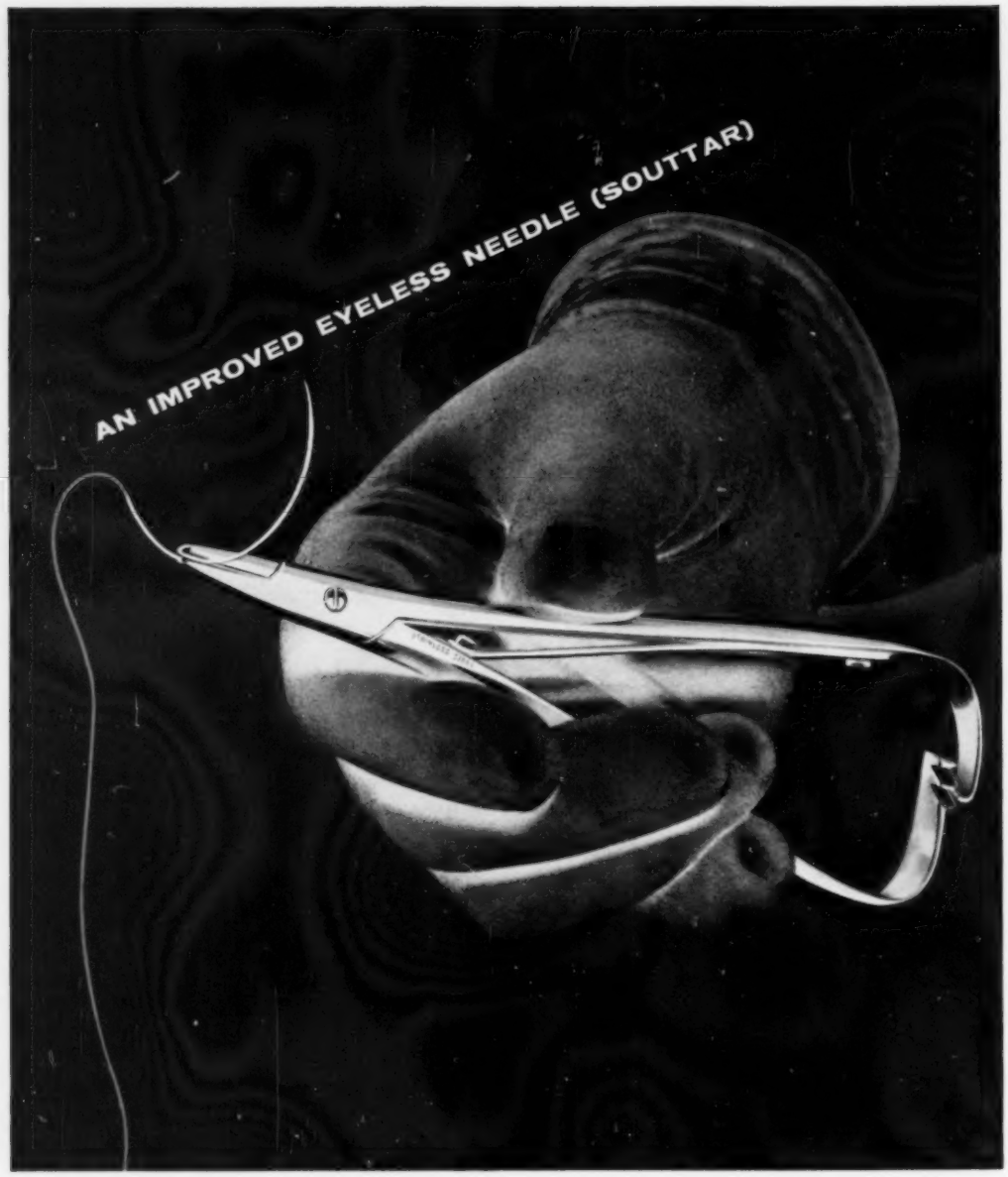

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THE
BRITISH JOURNAL OF SURGERY

VOL. XLVII MARCH, 1960 No. 205

THE SURGERY OF THE ŒSOPHAGUS*

BY SIR HENEAGE OGILVIE, LONDON

GEORGE GREY TURNER had two great interests in life, the History of Surgery and the School of Medicine of Newcastle upon Tyne.

When we consider the emergence of a school rather than of a gifted individual, we must seek a common stimulus that animated a number of individuals.

In many cases we find it in a national spirit. The surgeons of London, Edinburgh, Cardiff, and Dublin are aware that whatever they achieve will rebound to the credit of the race in whose capital they work. The surgeons of Belfast are urged even more strongly by the patriotism of the beleaguered fortress.

How then do we explain the fantastic virility and productivity of the Surgery of Newcastle, a city that has no capital status, indeed that was for centuries the home of border warfare between feudal communities? The answer is, I think, that the capacity of the human mind for unselfish devotion varies inversely with the size of the group that claims that loyalty. The citizens of Little Rock show a more passionate concern in the interests of Little Rock than in the welfare of the United States. The patriotism of a college exceeds that of a university. The spirit of a regiment will inspire an unselfish devotion exceeding the love of country.

It was said of one of the most famous Divisions in the Middle East, the 51st or Highland Division, that they feared only God and the 50th Division, the famous 'T and T' or Tyne and Tees Division, who were their most serious rivals. These 'Geordies' were a race apart. They were small, they were tough, and they talked to each other in a dialect that one was able to recognize only after some puzzlement as English. They were straight, in their thinking and their speech. They were loyal, to each other, to their officers, and to their regiment. They were brave in battle and uncomplaining when wounded or suffering, and they voiced their suffering, their complaints, and their needs in very few words. One came to love them, and one came to appreciate the deep affection and respect that they could inspire in their fellow Northumbrians, the love that was one of the motive powers in Grey Turner's life, and in his surgical achievements.

From this stock, and in these surroundings, a very definite school of surgery was bred: Hume, Rutherford Morrison, Grey Turner, Charles Saint, Hamilton Drummond, Clay, Willans, Norman Hodgson, Albert Hindmarsh. They had certain characteristics in common—physical toughness and an infinite capacity for hard work, sound common sense, good clinical judgement, a wonderful memory for cases, determination bred by many difficult jobs in the past, manual dexterity learned from constant operating, excellent craftsmanship, and no damned scientific nonsense. The inspiration common to all of them was the ever-present knowledge of the tremendous amount of work to be done, and of the need to do it "while it is yet day, for the night cometh when no man can work". They had a large number of beds in the Royal Infirmary, and waiting to fill them were their fellow townsmen, staunch loyal inarticulate men who were the best surgical material that any surgeon could hope for, bringing

FIG. 446.—George Grey Turner.

* Being the Grey Turner Lecture delivered October 27, 1959.

a body stunted and toughened by years of hard work and often privation, but indomitable courage, and a blind faith that could force the body to perform miracles of recovery.

I have tried to sketch the background from which the man we wish to honour sprang. I knew him only to the extent that the beginner knows the man who blazed the trail he is trying to follow. Grey Turner was a great figure in surgery at the time when I was struggling for the right to call myself a surgeon, a leader of surgical thought to whom I brought the members of my newly formed Travellers Club for guidance, a senior member of the governing body of the College of Surgeons when I had just been elected to the Council.

FIG. 447.—Group of House Officers at the Royal Victoria Infirmary, taken in 1899. Grey Turner is second from the right.

My memory of Grey Turner is of the man who knew everything, who probably knew more about surgeons, surgical history, and surgical methods than any man who had ever lived. At any meeting that I have attended, whatever the subject, it was Grey Turner's contribution that was the highlight of the occasion, the discussion that most people in the room had come to hear, the summary for which they must wait. He had no stereotyped approach. He talked as the spirit moved him from the unbelievably rich stores of his well-stocked memory. He could always give his own experience, some cases he had seen, the difficulties they had presented, and the way he had overcome them. If he had sufficient warning he could probably show a specimen or a slide and the photograph of the patient. If a question of the technique of an operation came up, Grey Turner had almost certainly done it himself, or he knew who had done it first and who had modified it and why, and how he had succeeded or failed. He never boasted. He never laid down the law or made claims for himself. He simply told what had happened; and the unvarnished tale very often served, though that was never his intention, to deflate the pretensions of a previous speaker.

Behind this serious and scholarly personality was the story of a life of hard work devoted to the study of surgery and the service of his native shires. He passed his finals in 1898 before he was 21, and had to wait awhile before his degree was conferred. He was appointed House Surgeon next year at the age of 22, when he worked so hard that his colleagues thought he would "burn himself out". It is said that on alternate nights he sat up all night copying out case notes, while the night nurse brought him hourly cups of tea.

But Turner's most remembered gift, his uncanny knowledge of global surgery and of the clinics of the world, he owed to the generosity of a relative, who sent him on a visit of several months to the clinics of Vienna. Billroth was recently dead, but his tradition was still vivid. Grey Turner in later years was never tired of recalling his experiences there. A few years later, when he was assistant surgeon at the Royal Victoria Infirmary, he returned to Vienna. The work of Hochenegg impressed him greatly, and did much to stimulate his already awakened interest in the surgery of the œsophagus.

The first of his many visits to America was in 1906, shortly after he was elected to the staff of the Victoria Infirmary. He went to attend the meeting of the British Medical Association in Toronto, and he took to visiting the chief surgical clinics in Canada and the United States. He was impressed by the personality and pioneer work of John B. Murphy, and by the great clinic of the brothers William and Charles Mayo.

In 1908 he married, and his wife thereafter became his constant encouragement, his loyal helper, and his companion on his journeys. In the same year he became a Territorial, and as a consequence

was called up for active service in 1914. In the last year of the war he was sent with an inter-allied surgical mission to America, to discuss the problems of war surgery with our newest allies. There he met many old friends in the States, and renewed his friendship with Pierre Duval of Paris and Bastianelli of Rome, who were also members of the delegation.

FIG. 448.—Professor Czerny of Heidelberg.

Foreign travel was now in his blood, and as one of the founders of the Moynihan Club and a staunch supporter and officer of the International Society of Surgery, he visited nearly every foreign surgical centre with his wife, and, in turn, welcomed nearly every surgeon of note to his clinic in Newcastle. 'Welcome' was no idle word with Grey Turner, for he not only opened his clinic, his operations, and his records to the visitor, but he delighted to show him the Border country of which he was so proud, its historic monuments, its folk-lore, and its music.

I have chosen the Surgery of the Œsophagus as the subject for my Grey Turner lecture because I think that he was more interested in that subject than any other in the last period of his career, and because when I became interested in it myself I found him before me in the battle, hacking at obstacles that were then insurmountable, and that were overcome too late for him to follow the path that now lay open. Yet by what he thought, by what he did, and by what he tried to do, he had a greater influence in shaping that attack on cancer of the gullet than any man in surgical history.

* * * * *

The history of œsophageal surgery is so closely linked with the name of Grey Turner that it is easier to discuss it in relation to him than to place him in one of the niches in the house that has risen round him.

The story starts in 1877, when the following preliminary report by Czerny, of Heidelberg, appeared in the *Centralblatt für Chirurgerie*:—*

> I have performed during the last few months a few new operations which, as far as I know, have not been performed on a living patient.
>
> 1. Resection of the Œsophagus.
>
> The patient (female 51 years old) had a circular carcinoma of the œsophagus which could not be passed with the aid of a bougie. The tumour occupied 6 cm. of the œsophagus in its whole thickness, which necessitated circular removal of 6 cm. of the œsophagus. The lower end of the œsophagus was sutured by interrupted stitches to the skin of the neck. The patient left the hospital cured.

Though Grey Turner was aware of Czerny's case, he refers in 1925 to the œsophagus as "a region where encouragement in the treatment of malignant disease has so far evaded many earnest workers". That surgical attack was in his mind is shown by his account of a patient who died after dilatation of an œsophageal stricture 17 months after the relief by gastrostomy. At post mortem the obstruction was found to be cancerous, but the disease was still localized.

In 1931 he gave the Bigelow lecture in Boston, his subject being "Some Experiences in the Surgery of the Œsophagus". He spoke of cases seen at Newcastle over more than thirty years, and after discussing non-malignant strictures of many kinds, he went on to talk of cancer of the œsophagus. He pointed out that in ten years nearly 16,000 people had died from the disease in Britain. He referred to the classical resection of Franz Torek, of New York, whose patient had just died at the age of 80, having survived a resection of the thoracic œsophagus for 14 years. He himself had seen no cases in the cervical œsophagus suitable for resection, but had resected two growths at the lower end without success. He considered that the transpleural route adopted by Torek and Eggers for the middle third of the œsophagus was too formidable and that the difficulty of restoring the œsophageal channel afterwards was too great. He described the relations and the blood-supply of the thoracic œsophagus in considerable detail, and told how he had tried mobilization of the œsophagus from the abdomen on eight occasions before attempting the collo-abdominal or 'pull-through' method of resection. He gave an account of the first case, in which he encountered unexpected difficulties and which died a week after the operation, and suggested that it might be possible to short-circuit the growth by making an anastomosis between the dilated œsophagus above and the fundus of the stomach below. This suggestion is now standard practice, but Grey Turner was never to carry it out himself.

Shortly after he returned from Boston Turner encountered the ideal case, a tough little Northumbrian miner aged 58, who gave an eight-weeks' history of dysphagia. After preliminary exploration

* The translation is by Dr. Richard Lewisohn of New York, Hon. F.R.C.S., who was Czerny's first assistant.

and gastrostomy, he removed the whole thoracic œsophagus on April 28, 1933, after freeing it from above and below by finger dissection. Two months later he reconstructed the œsophagus by making an antethoracic channel formed of a skin-tube above

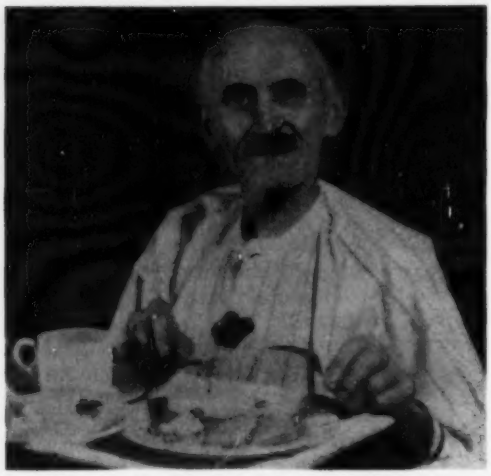

FIG. 449.—Grey Turner's first successful 'pull-through' operation.

and a jejunal loop below. The patient lived for 19 months, swallowing well, and died of uræmia "without obvious recurrence", but a small plaque removed from the stomach wall at post-mortem was found to be cancerous.

In 1934, when he had just taken the Chair at Hammersmith, he read a paper to the Royal Society of Medicine on 'Recent Advances in the Treatment of Cancer of the Œsophagus'. He described the technique of his 'pull-through' operation, and recounted 5 cases. The first lived a week, the second died on the night of operation, the third lived 19 months, the fourth (in which he removed the spleen and the whole of the stomach as well as the œsophagus) lived for 8 weeks and died of mediastinitis, the cervical œsophagus having sloughed. The fifth case died on the eleventh day of infection around the gastrostomy tube.

He chose 'Carcinoma of the Œsophagus' as the subject for his Bradshaw lecture in 1935, and discussed the pathological features of the disease and the anatomical problems of the surgical approach. He still considered that the collo-abdominal resection was the safest method in early and operable cases.

But his interest in the œsophagus was by no means limited to cancer. In the same year as his Bradshaw lecture, speaking at the Medical Society of London, he discussed achalasia, cicatricial stenosis, and impacted foreign bodies. In 1939 he wrote on non-malignant stenosis in the BRITISH JOURNAL OF SURGERY. He told of cases he had seen from the time he was a Registrar at Newcastle to the time when he was Professor at the Post-graduate School, and recounted a series of strictures of every type treated for the most part by bougies, some by œsophagogastrostomy, some by cardioplasty. He reported 6 œsophagogastric anastomoses not with the entire approval he had expressed formerly.

Towards the close of his active life he returned to the Royal Victoria Infirmary to give the Hume lectures on Injuries and Diseases of the Œsophagus. The book in which these lectures are published is one that every surgeon interested in the œsophagus should read and that every surgeon who knew the great man who gave them will treasure, for it enshrines his unrivalled experience and his profound wisdom, and it recalls his enthusiasm, his appearance, and the telling homely phrase in which he drove home his advice.

Grey Turner's years at the Post-graduate Hospital were devoted largely to the problems of œsophageal resection. I have examined at Hammersmith the records of 20 operations for cancer of the œsophagus. Of these 7 were resected with a primary mortality of 4 (or 57 per cent). Two were cured; a third, after gastrostomy, refused further surgery. Thirteen proved inoperable, of which 2 died. Adding to these cases the 5 previously reported, we have a total of 25 cases. Of these, 11 were resected, an operability rate of 44 per cent. Ten died, a mortality of 40 per cent, but of these 3 survived the operation for more than a week. Of the survivors, 1 lived 19 months—the famous first case—1 was alive a year after resection; 1 lived fourteen years and is still alive. I am glad to be able to show you his portrait taken at Norwich this month.

Grey Turner explored the abdomen on Jan. 18, 1945, and, finding no glands, he did a jejunostomy. At an œsophagoscopy a fortnight later, he found a fungating colloid carcinoma at the lower end. On Feb. 20 he opened the chest under intratracheal anæsthesia, divided the œsophagus below the root of the lung, and after opening the diaphragm he removed the lower end together with the proximal half of the stomach and the spleen. He closed the chest, and drew out the upper half of the œsophagus through a neck incision. He restored swallowing by making a skin tube on March 29, the lower end of which he joined by a jejunal loop to the stomach on May 1. The patient left hospital on July 7, 1945.

These numbers are small. Many young surgeons to-day resect more œsophaguses in the year than Grey Turner did in his life. But we must not forget that they are doing so because Grey Turner showed them the way. When Spencer Wells started to remove ovarian tumours, and when Ernest Miles started abdominoperineal resection of the rectum, their mortality was about 40 per cent; yet those who followed in their steps made these two operations among the safest in surgery.

Grey Turner's clinic was always the place to which any surgeon would repair if he wished to see œsophageal operations, to discuss œsophageal problems, or to seek advice on a case of dysphagia. He was always advancing, but he must advance at his own pace and in the direction he considered to be right. For very long he regarded the pleural cavity with the dread of his predecessors. Though he was one of the first to venture into the inferior mediastinum to anastomose the œsophagus to the stomach, he believed to the end that removal of the thoracic œsophagus implied a need to construct an antethoracic channel to

take its place. If a method worked, especially if he had learned it from a master, he was unwilling to replace it with something newer and possibly better, just as I, having driven motor-cars for fifty years am unwilling to change to one with an automatic gear-box.

He advocated gastro-œsophageal anastomosis and cardioplasty, and only towards the end was he prepared to admit that the functional results of these procedures are not always happy. He does not seem to have encountered reflux œsophagitis, and he never speaks of hiatus hernia. Herniation through the œsophageal hiatus in the diaphragm was known to Astley Cooper, but it is only since about 1945 that it has been recognized as a common complaint. The importance of reflux œsophagitis was not recognized till the paper by Barrett and Franklin in 1949.

His personal contribution was above all an infinite capacity for taking pains. He demonstrated time and again that he could, by patience and perseverance, find a channel through even the longest and tightest stricture, bring it by gradual dilatation to a diameter where the patient could pass bougies himself, and eventually restore normal swallowing. It is doubtful whether any present-day surgeon has inherited his skill; certainly none has his patience or gets his results.

In cancer he taught us two things: the immense importance of early diagnosis, and the concept of the thoracic œsophagus lying in a loose bursal sheath. Above all he taught courage, determination, and persistence, and he communicated these qualities to those who worked with and for him. With his encouragement his pupil Dick Franklin performed the first operation in Britain for the repair of congenital atresia in the newborn. In his unit, and under his direction, Franklin started the series of resections and short-circuits that culminated in his publication with Shipman of 150 cases in 1952.

* * * * *

The history of œsophageal surgery is that of a rocket; first the application of a match to a fuse, after an interval a sputter of flame, then after another pause a trail of fire going up into the darkness and ending in a blaze of light and a constellation of stars. The match was applied by Czerny in 1877, the sputter was Torek's case in 1913, the rocket started to go up in 1933 with Grey Turner's first case, and we are now looking up at stars too numerous to count.

All pioneers are optimists. Czerny did not cure his patient, but he lit a fuse, and for the next thirty years it smouldered, chiefly in Germany, France, and Switzerland. Sauerbruch tried to design a differential pressure chamber, and in 1910 he had performed 10 resections of the cardia; Kuttner had done 6, Mikulicz 5, Tuffier 3, and several others one or two each. All these patients died, but one of Sauerbruch's cases survived for two weeks. Meanwhile British surgeons were also at work. Arthur Evans resected a tumour of the cervical œsophagus and cricoid region in 1909. The patient was alive

FIG. 450.—Grey Turner's last œsophageal resection (F. F.). Resection of carcinoma of lower end of œsophagus at the Post-graduate School, February, 1945.

FIG. 451.—F. F., aged 67 years. Fourteen and a half years later. Photograph taken at Norwich in October, 1959.

and well 23 years later. Arbuthnot Lane excised a cancer of the cervical œsophagus in 1911 and replaced the defect by a skin-flap, using a method later associated with the names of Trotter and Wookey.

However, in the first thirty years of œsophageal surgery surgeons were less concerned with the hopeless task of excising cancer than with the more urgent one of providing a by-pass for irremovable obstructions. In 1904 Gluck and Perthes used a tube to carry food from an œsophageal fistula in the neck to a gastrostomy opening. In 1907 Roux, of Lausanne, joined a loop of jejunum carrying its blood-supply to the stomach below, and brought out the upper end over the chest. Roux fed his patient with a tube, but later Herzen and Kocher repeated his jejunal loop, joining the upper end to the œsophagus in the neck. In the same year Bircher attempted to make a skin-tube and in 1911 Kelling tried to use the transverse colon. In 1912 Frangenhein made a by-pass of the skin above and a jejunal loop below that lasted a year. In 1913 Meyer suggested an operation to join the œsophagus in the thorax to a tube formed from the greater curvature of the stomach, a suggestion that was twenty years before its time.

The stage was now set for the next phase. In 1913 Torek, of New York, and a little later Zaaijer, of Leyden, resected the thoracic œsophagus by the transpleural route. There is no doubt that the advance that made these operations possible was the invention by Melzer of intratracheal anæsthesia in 1911. Both surgeons closed the lower end, and brought the upper end out in the neck and joined œsophagus to stomach by a rubber tube. Torek's patient lived for 14 years. From then on advance proceeded steadily, but slowly at first. Eggers had two successes by the Torek method in 1925 and 1930, and Hedblom one in cancer of the cardia. Lilienthal in 1921 resected the thoracic œsophagus by the difficult posterior extrapleural route, and replaced the gap with a skin-flap.

Grey Turner's successful case came in 1933, and in the same year Ohsawa in Japan reported 18 resections of the lower end of the œsophagus with 8 survivors. From then the pace increased. In 1936 King, of Melbourne, published another successful removal of the thoracic œsophagus. In 1938 Garlock, of New York, reported 3 successful Torek resections. In the same year, 1938, Adams and Phemister reported the first transpleural resection of the œsophagus followed by œsophagogastrostomy, an operation that may be said to have inaugurated the present phase of œsophageal surgery.

The present phase can be dated from the second world war. The second war, like the first, gave young surgeons the opportunity to undertake major surgery and to make important decisions when their hands and their brains were at their best, at a time when the scientists of the world were developing and perfecting new drugs, new instruments, and new methods. In September, 1939, surgeons still regarded cancer of the œsophagus as a wellnigh hopeless problem. The fear of pneumothorax forbade any entry into the thorax except by extrapleural routes. Torek's resection was a solitary triumph, and any future surgeon could hope for success only by repeating his method. The lower end of the œsophagus must be closed, the upper end must be brought to the surface, and a new antethoracic channel must be constructed somehow.

In 1938 two notable contributions pointed the way to further advances. The first, the publication of Garlock's cases, showed that the risks of œsophageal resection were no longer prohibitive. The second, Adams and Phemister's case, suggested the direction of future development. At first Garlock in America and Grey Turner in England continued to do Torek resections, and Yudin in Russia perfected Roux's jejunal by-pass. But towards the close of the war the two principles of transpleural resection and immediate restoration of continuity were being advocated by Garlock and Sweet in the United States and by Ivor Lewis in Britain.

With the cessation of hostilities in Europe, the thoracic units which had been developed and trained in the Forces and in the Emergency Medical Service were released for civilian work. The surgeons had learned to combat shock and infection, the anæsthetists had developed closed respiration anæsthesia and the use of relaxants. These skilled teams formed new units in university surgical departments, or were grafted on to others already in existence, and they now turned their skill to the pulmonary surgery, the cardiac surgery, and the œsophageal surgery of peace. One-stage œsophagectomy with œsophagogastrostomy became standard and the Torek œsophagectomy, gastrostomy, cervical œsophagostomy, and the extrathoracic œsophagoplasty were abandoned. The mortality fell from 90 per cent in the early war years to about 30 per cent now.

Since I cannot epitomize present-day views on technique, I give a summary of my own experience and opinion. At the start of the present phase I was writing *Recent Advances in Surgery*, studying the anatomy of the œsophagus, and practising possible methods of attack in the dissecting room. In 1936, two years before Adams and Phemister's paper, I published in the BRITISH JOURNAL OF SURGERY an operation in which I had joined the œsophagus to the stomach over a tube. My views have been greatly influenced by two men: by Grey Turner, who pointed out that the lower few inches of the œsophagus can be freed by blunt dissection from the abdomen; and by Ivor Lewis, who showed that only from the right side can the whole length of the thoracic œsophagus be dissected under direct vision. These views are as follows:—

1. Gastrostomy has no longer a place in œsophageal surgery, except for resting the œsophagus in the acute phase of corrosive poisoning.

2. The approach to the œsophagus is through the cavity in which the œsophagus lies—the chest. The hiatus is well up in the thorax, near the summit of the diaphragmatic dome; the approach to it from the thorax is across an empty space, the approach from the abdomen is an exploration into a deep hole overhung by costal margins, filled with gaseous viscera that must be kept out of the way with packs and retractors, and obstructed by the left lobe of the liver that must be mobilized before it can be retracted. I always use the thoracic approach for the repair of hiatus hernia and for extramucous myotomy in achalasia. I have watched thoracic

colleagues do the same operations from below and have marvelled that excellent surgeons should take so much trouble to get themselves into such a needless mess.

3. Heller's operation is the only successful treatment for achalasia and it is always successful when can, and should, be divided between ligatures because with its division the bundle opens out, the œsophagus lies exposed throughout its length, and it can be detached by clean knife dissection from the aortic arch and the left bronchus.

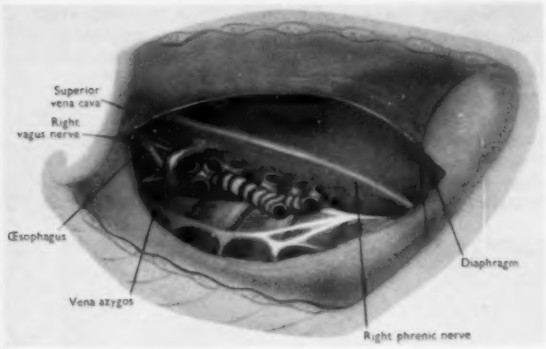

FIG. 452.—The right mediastinum after removal of the right lung. The pleura has been turned aside to show the structures beneath it.

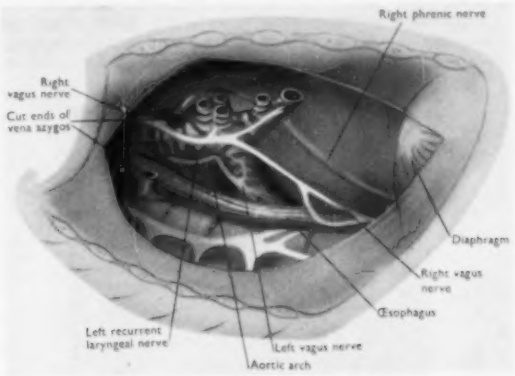

FIG. 453.—The right mediastinum, after division of the vena azygos major, and retraction of the structures that cover the œsophagus. (Œsophageal branches of the right vagus have been cut to simplify the dissection.)

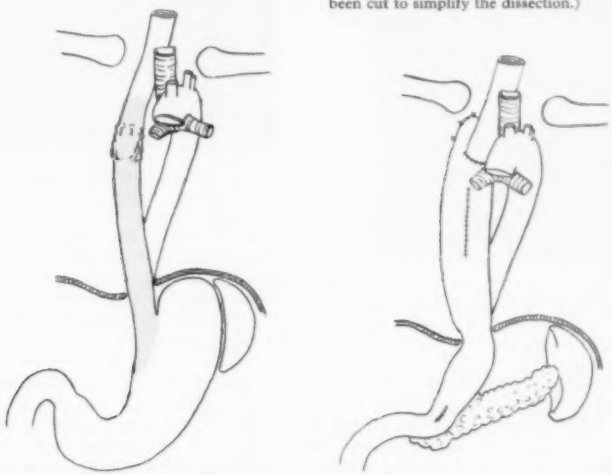

FIG. 454.—Diagram to illustrate the standard operation for resection of a carcinoma of the middle third of the œsophagus.

performed through the thorax. Cardioplasty is always disastrous, and the nearer any œsophagogastric anastomosis is to the cardia, the more likely is it to lead to œsophagitis and eventually to stenosis. Reflux œsophagitis is a matter of differential pressure between the stomach and the œsophagus, and the higher the anastomosis is in the chest and the more intragastric pressure is abolished by pyloroplasty, the less likely it is to occur.

4. The thoracic œsophagus should be approached from the right side. The mediastinum can be likened to a bunch of asparagus tied at the two ends and in the middle. The ties in the middle are the vena azygos major on the right, and the aortic arch on the left. The aortic arch cannot be divided, and should not be handled roughly. The vena azygos

Blunt dissection and finger dissection of the œsophagus should be reduced to a minimum, and if possible the œsophagus should never be pulled out from under the aortic arch and brought down the other side. To mobilize the aortic arch by dividing the upper intercostals so that it can be pulled out of the way of the œsophagus is to invite paraplegia owing to the interference with the spinal vessels.

5. The chief danger of œsophageal surgery is post-operative chest complications. The diaphragm should never be divided unnecessarily, and it should never be paralysed.

6. There is no place in civilian surgery for the abdominothoracic incision, except for the operations of portocaval anastomosis and hepatic lobectomy.

In these operations the liver must be turned up into the chest to allow access to its visceral surface without vigorous retraction. All other abdominothoracic operations are partly abdominal and partly thoracic. Parts of them are better performed from below, parts from above, but no parts are better done from the side.

The abdominothoracic incision gives excellent access—it might even be termed 'indecent exposure'—but at a price that might be considered excessive.

FIG. 455.—A. M., aged 69 years. An ulcerating carcinoma 2½ in. long, with its upper border level with the top of the aortic arch, was resected by the method described in the text by Mr. T. A. Ogilvie of Colchester in May, 1954. Portrait taken in October, 1959.

It cuts right through the rim of the thoracic cage, and this costochondral ring is as important in the upper framework of the abdominal wall as the pelvic ring is in the lower. It provides the origin of the diaphragm above and of the abdominal muscles below. Once divided it never unites firmly again. Bone can unite by bone, but cartilage never unites by cartilage or even by good fibrous tissue.

That a patient can keep reasonably well during a four-hour operation with the thoracic cage split open and the thoracic and abdominal cavities thrown into one is no argument in favour of that approach. It is a compliment to Nature's power to cope with adverse circumstances, rather than to the surgeon who thinks he knows better.

7. The standard operation for œsophageal resection implies an œsophagogastric anastomosis, which should be done in the right chest. Properly mobilized the fundus of the stomach can be brought as high as the cricoid without interfering with its blood-supply, and without cutting the diaphragm.

The abdominal part of the operation is done through a midline incision extending from the xiphisternum to the umbilicus. The great omentum is divided between ligatures distal to the gastro-epiploic arch, and the division extended on the right to within 2 in. of the pylorus. On the left it is continued to the splenic flexure, and then the short splenic arteries are divided till the diaphragm is reached on the left of the œsophagus. The spleen and pancreas are not disturbed. The stomach is then turned up, the left gastric artery is ligatured at its origin, and the lesser omentum is divided until the diaphragm is reached to the right of the œsophagus. The left lobe of the liver is now displaced, and the stomach is drawn down while the peritoneum in front of the hiatus is divided. The so-called œsophagophrenic ligaments are structures of no importance. The thoracic œsophagus can now be pulled down for a couple of inches as in vagotomy,

FIG. 456.—A. M., October, 1959. The scars are now invisible, but the thoracic one, which is over the 6th rib, has been inked in for the purpose of the photograph.

but the object now is not to isolate the vagi, but to isolate vagi and œsophagus together in the bursal space in which they lie. This is easily done by the finger, or a small swab on a holder passed through the hiatus, and swept around while the œsophagus is pulled down with the other hand.

Before passing to the chest, the pyloric sphincter is divided without opening the lumen, and the peritoneum lateral to the duodenum is divided to allow the gastroduodenal artery, from which the blood-supply of the displaced stomach will be derived, to swing up without tension. The abdomen is closed with all-layer sutures of swordfish nylon and the patient, who has lost no blood and should still be fighting fit, is handed over to the anæsthetist, who turns him into the left lateral position while the surgeon changes his gown and his gloves.

On his return the surgeon opens the right pleural cavity at the appropriate level, usually in the bed of the fifth rib. He incises the mediastinal pleura freely, divides the vena azygos, and proceeds to identify the growth, to mobilize the œsophagus above and below it, and to search for distant metastases. The involved region is dissected free with the knife with the greatest amount of surrounding tissues that anatomical limits will allow. The boats have not yet been burned, the blood-supply of the œsophagus and the stomach have not yet been jeopardized, and if the operation proves impracticable even now no irreparable harm has been done.

If the green light shines, the incision in the mediastinal pleura is continued downwards until it opens into the posterior mediastinum already prepared from below. The mobilized stomach can then be pulled into the chest through the uncut hiatus. The cardiac end of the œsophagus is excised with a wide ellipse of lesser curvature, for the longitudinal fibres of the œsophagus continue into the lesser curve of the stomach and form an inextensible band that prevents mobilization of the fundus.

The fundus, thus freed, is fixed by interrupted sutures to the endothoracic fascia well above the lower limit of intended œsophageal division. The œsophagus is cut across well above the growth, leaving a little collar of mucous membrane protruding, and implanted in a circular hole cut from the anterior wall of the fundus with two rows of sutures. The thoracotomy incision is closed.

8. The problem is different when the growth is at the cardia, or in the lowest 2 in. of the œsophagus, for radical surgery demands the removal of the glands accompanying the left gastric and splenic artery, that is the excision must include the lesser curvature of the stomach, the spleen, and half the pancreas, and the œsophagus must be anastomosed to a tube of the greater curve of the stomach or to a Roux loop of the jejunum. The approach to the lower œsophagus is through the left chest and the anastomosis should be done in the left hemithorax, but the abdominal operation and the intrathoracic anastomoses are separate problems.

Though my subject is an historical one, I shall not attempt to give present-day statistics. Moynihan said that statistics can prove anything, even the truth, but they do not give the truth unless they are collected and analysed by men who took no part in the work and who are indifferent to the result. Surgeons' own figures are either too good to be true, or too true to be good. Grey Turner always spoke the truth. He was never a 'hundred-per-center', like the surgeon to whom Charlie Mayo remarked: "Your figures for thyroids are almost as good as ours for Cæsarean section. We start with one patient and finish with two."

Throughout the campaign in the Western Desert the mortality of the Field Surgical Units for abdominal wounds was 30 per cent, a figure of which I was, and still am, very proud. The consulting surgeon of another army claimed a mortality of 15 per cent. I noticed that in a recent action his surgeons had done about a third of the operations that ours had, and that his early post-operative mortality was negligible, while 42 per cent of our deaths were in the first 24 hours. When I put this to him, he replied, "I have learned to know when a man is going to die, and I do not want to waste my boys' time". In the Eighth Army time was nothing, risk was nothing, and statistics were nothing, if there was a chance that a life might be saved. That should be the outlook of the œsophageal surgeon.

I have put together the recent figures of surgeons whose work I know to be good. John Garlock and Richard Sweet in America; Allison, Barrett, Belsey, Brain, Cleland, Collis, Cox, Franklin, Edwards, Lewis, Logan, Miller, Mullard, Holmes Sellors, and Vincent Thompson in Britain. The grand total of resections for cancer is 1371, of which 337 died, giving a hospital mortality of 24·6 per cent. The majority of surgical records give a similar figure for deaths, but in small series in which the element of luck can be the dominant factor, it can vary between 15 and 50 per cent.

To estimate the cure rate is impossible, for surgeons are rightly concerned with the need to encourage sufferers and the doctors who advise them,

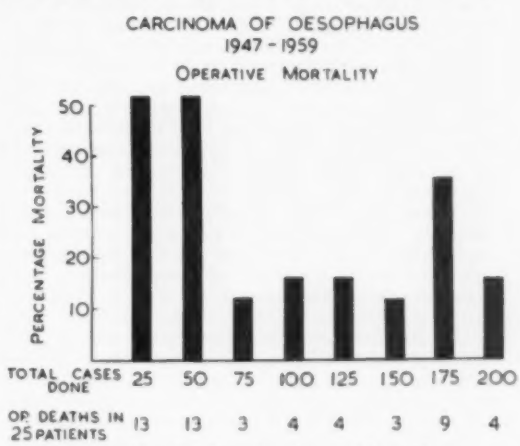

FIG. 457.—Mortality of resection of cancer of the œsophagus, analysed in consecutive series of 25 cases each. (Chart kindly provided by J. Leigh Collis, Esq.)

and they so classify their results in groups according to age, site, and operability that it is difficult to discover the truth. I can only quote from personal letters written to me by three leaders. Belsey writes: "The commonest single cause of death was cardiovascular disease and such catastrophes as pulmonary embolism. The age of the patient plays some part in determining the mortality rate. In 160 patients under 70, the average mortality was 15 per cent, in 60 patients over 70 the mortality rate was 70 per cent." Lewis writes: "The mortality is still round 25 per cent. The most important single factor in mortality is the age of the patient. The longest survival for the middle third is just over three years, but I have had half a dozen of the lower end living five years or more." Collis writes: "The upper end of the œsophagus is not treated by surgical methods. About half the cases for whom hospital admission is arranged have a radical resection. In the case of patients in whom the growth was only explored it is certainly not true that no benefit was conferred. In a few cases a by-pass was done, but in the main my palliative manœuvre is to pass a tube through the growth area from above, and for one of my assistants to guide it through and see that it is properly placed. This he does through the open chest. It is very rare for any patient with carcinoma of the œsophagus not to be sent out of hospital swallowing.

"We have not felt it reasonable to take a five-year survival, as there are so few in this category. Taking a two-year survival, about a third are still alive at the end of two years."

Radiotherapy with modern high-voltage machines can often palliate even advanced cases, and restore

swallowing for a time, but the cures are distressingly low. Smithers in 229 cases treated had a two-year survival of only 9 per cent, and Mustard in 125 patients a two-year survival of 8 per cent. Windeyer, in a personal communication, says: "I am afraid that our experience in carcinoma of the œsophagus tells a doleful story. There are only one or two who have gone more than a year or thereabouts. I think that this is the general opinion of all radiotherapists who have been treating these cases. The cause of failure in our cases has, for the most part, been that the patient developed a recurrence above and below the area treated and a number have developed metastatic lesions that one had not previously seen with carcinoma of the œsophagus." The most hopeful feature is that the best results of radiotherapy are in the upper third, the section in which the results of resection are the worst.

The picture I have drawn is in some ways depressing. The mortality of œsophageal resection is high, but taken in conjunction with the age and debility of the patients, with the frequency of accompanying cardiovascular disease, and with the appalling immediate prospects that confront them it is not prohibitive. The cure rate is poor. But the alleviation rate in those that survive the operation is nearly 100 per cent. The sufferers are relieved from the fear of starvation or, still worse, of thirst. Though the disease may progress, dysphagia does not recur and they die of a general cachexia without any intolerable pain, swallowing to the end.

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THE INNERVATION OF THE CARDIA AND LOWER ŒSOPHAGUS IN MAN

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IN spite of the fact that recognition of abnormalities of the œsophagus and gastro-œsophageal junction has advanced rapidly in the last fifteen years, the physiology of this part of the alimentary tract is still incompletely understood.

The extensive literature on œsophageal function has recently been ably reviewed by Ingelfinger (1958). Much of this work is concerned with various animals, and though of considerable interest may not be relevant to human beings because there are widespread differences between species in the type of muscle in the wall of the œsophagus and in its innervation. In this paper we have confined ourselves to the human œsophagus.

The literature contains considerable information on the sequence of events in the human œsophagus following deglutition. These have been studied largely by radiography and by recording intraluminal œsophageal pressures by different methods. There is much less information on the part played by

the autonomic nervous system in œsophageal movements.

The assessment of autonomic control requires a reliable method of alteration of autonomic balance. In man alteration of balance has been produced by denervation of the œsophagus and by the administration of drugs which interfere with some part of the autonomic system.

In practice, denervation of the œsophagus may result from vagotomy or thoracolumbar sympathectomy. Transient dysphagia and delay in the cardia occasionally occur following vagotomy for peptic ulceration, and failure of relaxation has been demonstrated radiologically (Freidburg, 1950; Grimson, Baylin, and others, 1947). In view of the low level of section of the nerve in this operation no conclusions can be drawn with regard to the effect of total interruption of the vagal nerve-supply. In 3 cases (personal observation) in which high unilateral thoracic vagotomy has been performed during pneumonectomy, a condition clinically and radiologically indistinguishable from achalasia of the cardia has occurred. In one of these the remaining vagus may have been involved by recurrence of the growth in the mediastinum. No reports of œsophageal dysfunction following bilateral thoracolumbar sympathectomy for hypertension have been found in the literature. Among 60 such cases reviewed (Ellis, 1955) no difficulty in swallowing was noted. Grimson, Reeves, Trent, and Wilson (1946) found no radiological or pressure changes in the human œsophagus after thoracolumbar sympathectomy.

Such observations suggest that the main motor innervation to the œsophagus is mediated by the vagus and that in addition the vagus may also be in some way concerned with relaxation of the cardia. However, they lack precision, and because of the extensive nervous supply of the œsophagus complete interruption of all pathways is unlikely to have occurred even with thoracolumbar sympathectomy. In addition, such observations are for obvious reasons incapable of being repeated.

There is now a considerable range of drugs which interfere with various aspects of autonomic function and it may be reasonable to suppose that by observing the effects of these drugs on œsophageal function it would be possible to arrive at some conclusion concerning the relative parts played by the sympathetic and parasympathetic nervous systems.

In man cholinergic drugs have little action on normal œsophageal activity. In some cases (Sleisenger, Steinberg, and Almy, 1953) no effects were recorded, while in others intraluminal pressure (Lambling, Bader, and Rivoal, 1956) and balloon-induced peristalsis (Puppel, 1950) indicated increased activity. Adrenergic drugs in the maximum tolerance of doses have been without effect on the normal œsophagus (Schmidt, 1939; Baylis, Kauntze, and Trounce, 1955). Atropine reduced balloon-induced peristalsis (Baylis and others, 1955; Puppel, 1950), though its effects were not found to be consistent by some (Lambling and others, 1956; Schmidt, 1939). Methantheline has a greater effect on intraluminal pressure measurements (Ingelfinger, Kramer, and Sanchez, 1954), producing marked diminution in propulsive movements and well-marked radiological delay and retention. Assessed radiologically (Sleisenger and others, 1953) adrenergic blocking drugs and ganglion blocking drugs were found to be without effect. Information concerning the effect of autonomic drugs on the normal cardia is limited. Altered œsophageal motility itself is likely to affect the tone of the cardia, and when drug-induced the distinction between the effect on the cardia of these drugs and of disordered œsophageal movement is impossible. Œsophageal delay following the administration of anticholinergic drugs has been thought to indicate increased tone in the cardia (Ingelfinger and others, 1954), but subsequent intraluminal pressure measurements in this zone suggest the opposite (Ingelfinger, 1958). Adrenaline and noradrenaline (Sleisenger and others, 1953) did not alter the function of the cardia in normal persons and adrenergic blocking agents had no effect. From these results it is clear that no definite pattern of autonomic innervation has been distinguished. The major difficulty in producing alteration of autonomic balance by the administration of drugs is that it is impossible to give with safety sufficiently large doses to produce clear-cut effects. Hitherto the methods of assessment of alteration of autonomic activity in the human œsophagus have been direct observation, radiological studies, and intraluminal pressure measurements (Butin, Olsen, Moersch, and Code, 1953; Code, Creamer, Schlegel, Olsen, and others, 1959; Fyke and Code, 1955). While these methods provide information about the activity of the œsophagus as a whole, they fail to provide discrete information on contraction and relaxation of the longitudinal and transverse muscle. In the method about to be described the difficulties of drug intolerance have been overcome and the method of recording provides accurate information on contraction and relaxation of both longitudinal and circular muscle. There is no limit on repetition of the method.

METHOD

At thoracotomy strips of normal œsophageal muscle were removed from the extreme lower end of the œsophagus (the cardia) and from a site well above the cardia immediately below the hilum of the lung. The strips measured about 2½ cm. in the long axis of the muscle-fibres and about 0·5 cm. across. The longitudinal and transverse (circular) muscle coats were easily separated. From the cardia ten strips of longitudinal muscle and six strips of transverse muscle were obtained. From above the cardia, ten strips of longitudinal muscle and eight strips of transverse muscle were studied. Immediately after removal the muscle strips were suspended in aerated tyrode solution at 37° C. in a 50-ml. bath. The effect of a drug was assessed by adding it to the bath and by recording the response through a lever writing on a smoked drum. Before the addition of a further drug the muscle strip was washed repeatedly and allowed to regain its former state of tone indicated by the lever reaching the base line. Each muscle strip was further investigated by assessment of its response to electrical stimulation. Silver electrodes were applied to the ends of the suspended strips. Immediately before stimulation the level of the tyrode was lowered until the muscle and electrodes were completely uncovered. The strip was then stimulated along its longitudinal axis and the response recorded on a revolving smoked drum.

The stimulator provided simple rectangular voltage pulses of variable duration (0·01 m.sec. to 300 m.sec.) and intensity (0 to 200 volts). Its output impedance was 300 ohms. In preliminary experiments the current wave form was observed simultaneously with the voltage wave form by means of a double-beam cathode-ray oscilloscope. It was confirmed that the current wave form was also substantially rectangular except for the usual small spike of relatively short duration (associated with the capacitative component of the impedance between the electrodes) at the rising edge of the pulse. The resistance of the electrodes and specimen ranged from 2000 to 4000 ohms.

The threshold voltage required for stimulation was determined by increasing the amplitude of the pulse-waves in steps until a muscular response was recorded. The tyrode level was then raised and the muscle allowed to rest before another measurement of threshold for a pulse of longer duration.

In all these experiments 5 pulses per sec. for 5 sec. were used for stimulation.

RESULTS

Response to Drugs.—In *Tables I–III* the response of the muscle strips to autonomic drugs is indicated.

1. *Cholinergic Drugs* (*Table I*).—The addition of acetylcholine produced immediate strong contraction in all muscle strips. This contraction was prevented by adding atropine before acetylcholine, but succinyl choline had no such blocking effect. The cholinergic response of the circular muscle at the cardia and its prevention by atropine is shown in *Fig.* 458 A, B, C.

2. *Adrenergic Drugs* (*Table II*).—The response to adrenergic drugs was more complicated. Adrenaline and noradrenaline were found to have the same effects. Transverse strips of muscle from the cardia contracted after the addition of these drugs (*Fig.* 458 D, E). If phentolamine was added to the bath previously, adrenergic drugs (*Fig.* 458 F) constantly produced relaxation. These results could be obtained after atropinization of the preparation had abolished cholinergic activity and must therefore be due to a direct effect on the muscle.

While the adrenergic responses of transverse muscle from the cardia were constant, muscle strips from remaining sites responded variably. Longitudinal muscle from above the cardia remained inert. Transverse muscle from this area and longitudinal muscle from the cardia responded weakly, sometimes by contracting and sometimes by relaxing. After phentolamine relaxation only occurred.

3. *Nicotine* (*Table III*).—Above the cardia all muscle strips contracted following the addition of nicotine. This response was augmented by eserine and sometimes only occurred after the preparation had been eserinized. It was consistently blocked by atropine.

Transverse strips of muscle from the cardia responded quite differently, nicotine producing relaxation (*Fig.* 459 A). After eserination this relaxation still occurred, but was followed by contraction (*Fig.*

Table I.—Response of Muscle Strips to Cholinergic Drugs

	Transverse Cardia	*Longitudinal Cardia*	*Transverse above Cardia*	*Longitudinal above Cardia*
Acetylcholine 50 μg.	+ ↑	+ ↑	+ ↑	+ ↑
Atropine 100 μg.	O	O	O	O
+ Acetylcholine 50 μg.	Block	Block	Block	Block
Succinylcholine 2·0 mg.	+ ↑	+ ↑	+ ↑	+ ↑
+ Acetylcholine 50 μg.	(No block)	(No block)	(No block)	(No block)

+ ↑ = Responds by contracting; O = No response.

Table II.—Response of Muscle Strips to Adrenergic Drugs

	Transverse Cardia	*Longitudinal Cardia*	*Transverse above Cardia*	*Longitudinal above Cardia*
Noradrenaline 100 μg. or Adrenaline 100 μg.	+ ↑	+ ↑ or + ↓	+ ↑ or + ↓	O
Phentolamine 0·5 mg. + Noradrenaline 100 μg.	+ ↓	+ ↓	+ ↓	O

+ ↑ = Responds by contracting; + ↓ = Responds by relaxing; O = No response.

Table III.—The Pattern of Innervation of Muscle Strips when stimulated by Nicotine

	Transverse Cardia	*Longitudinal Cardia*	*Transverse above Cardia*	*Longitudinal above Cardia*
Nicotine 450 μg.	+ ↓	O + ↑	O + ↑	O + ↑
Atropine 100 μg. + Nicotine 450 μg.	+ ↓	O (Block) or + ↓	O Block	O Block
Eserine 100 μg. + Nicotine 450 μg.	+ ↓ ↑	+ ↑ (increased)	+ ↑ (increased)	+ ↑ (increased)
Eserine 100 μg. + Atropine 100 μg. + Nicotine 450 μg.	+ ↓	O Block	O Block	O Block

+ ↑ = Responds by contracting; + ↓ = Responds by relaxing; + ↓ ↑ = Relaxation followed by contraction; O = No response.

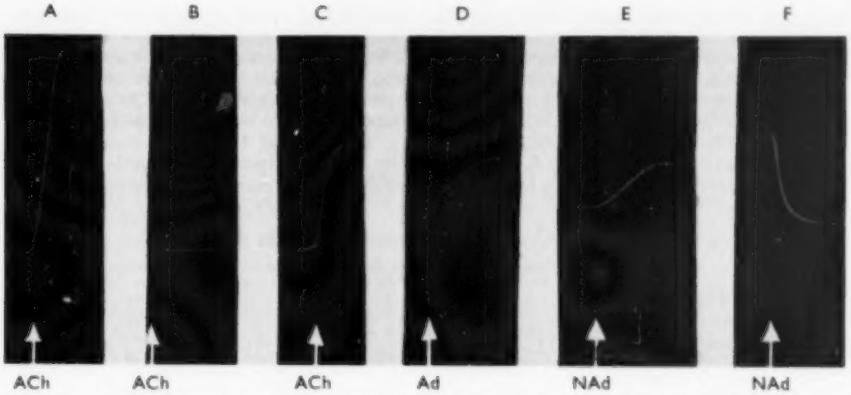

FIG. 458.—The response of the circular muscle at the cardia to cholinergic and adrenergic drugs. Drugs added to 50-ml. tyrode bath at 37° C. Drum speed 0·064 mm. sec.
A, ACh = Acetylcholine 50 μg. B, Atropine 100 μg. followed by acetylcholine 50 μg. C, ACh = Acetylcholine 50 μg. D, Ad = Adrenaline 100 μg. E, NAd = Noradrenaline 100 μg. F, NAd = Phentolamine 0·5 mg., followed by noradrenaline 100 μg.

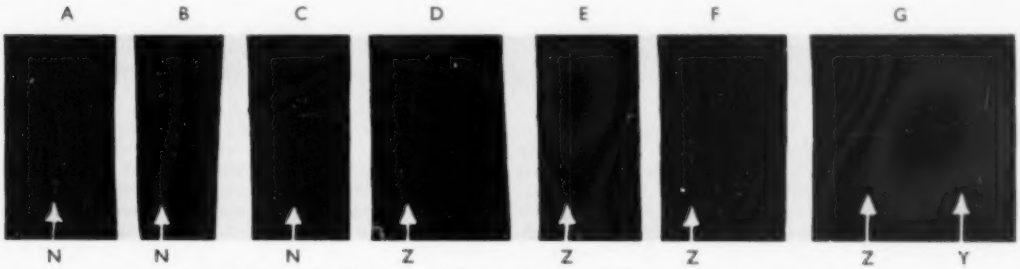

FIG. 459.—The pattern of innervation of circular muscle at the cardia. A, B, C: Drugs added to 50 ml. bath at 37° C. Drum speed 0·064 mm. sec. D, E, F, G: Electrical stimulation at 5 pulse-waves per sec. for 5 sec. at 200 volts. Drum speed 0·296 mm. sec.
A, *N* = Nicotine 450 μg. B, *N* = Eserine 100 μg. followed by nicotine 450 μg. C, *N* = Eserine 100 μg. followed by atropine 100 μg. followed by nicotine 450 μg. D, *Z* = Stimulation of nerves by short-duration (0·03 m.sec.) pulse-waves. E, *Z* = Short-duration pulse-wave stimulation after eserine 100 μg. F, *Z* = Short-duration pulse-wave stimulation (0·03 m.sec.) after eserine 100 μg. and atropine 100 μg. G, Preparation cooled to 14° C.: *Z* = Short-duration pulse-wave stimulation (0·03 m.sec.); *Y* = Long-duration pulse-wave stimulation (0·3 m.sec.).

459 B). The latter is blocked by atropine (*Fig.* 459 C). Longitudinal muscle at the cardia responds to nicotine by contracting. This is augmented by eserine, but after atropine nicotine sometimes produces a slight relaxation.

Response to Electrical Stimulation (*Table IV*).—Electrical stimulation may be used to evoke a response in muscle strips in two ways. Short-duration pulses (0·03 m.sec.) stimulate the nerve elements only, while long-duration waves (0·3 m.sec.) stimulate the muscle-fibres directly.

Using long-duration pulse-waves (i.e., stimulating muscle-fibres directly) contraction occurred in all muscle strips. In *Fig.* 460 this effect is shown in circular muscle above the cardia (A, *Y*) and circular muscle from the cardia (B, *Y*). The addition of atropine did not effect this response.

Using short-duration pulse-waves (i.e., stimulating the nerves) contraction still occurred in all sites except in circular muscle at the cardia, where (as with nicotine) relaxation occurred. In *Fig.* 460 the response of circular muscle above the cardia (A, *Z*) is compared with that of the circular muscle at the cardia (B, *Z*).

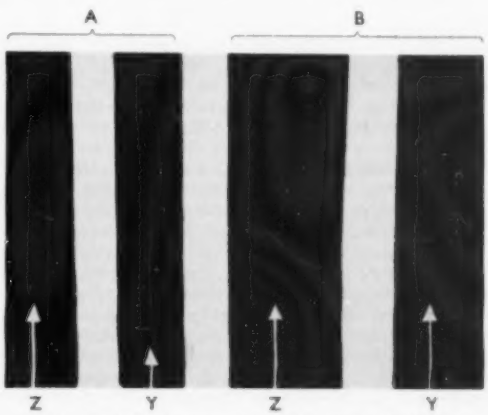

FIG. 460.—The response of circular muscle to electrical stimulation. Drum speed 0·296 mm. sec. All strips stimulated at the rate of 5 pulse-waves/sec. for 5 sec. at 200 volts.
A, Circular muscle above the cardia: *Z* = Short-duration pulse-wave (0·03 m.sec.); *Y* = Long-duration pulse-wave (0·3 m.sec.). B, Circular muscle at the cardia: *Z* = Short-duration pulse-wave (0·03 m.sec.); *Y* = Long-duration pulse-wave (0·3 m.sec.).

In *Fig.* 459 D relaxation of circular muscle at the cardia or stimulation by short-duration pulse-waves is shown again. If stimulation is repeated after the addition of eserine (*Fig.* 459 E) the relaxation is sometimes followed by contraction. The latter is mediated through a cholinergic mechanism, since (*Fig.* 459 F) the addition of atropine leads to its disappearance.

It will be seen that nicotine (*Fig.* 459 A, B, C) and short-duration pulse-waves (*Fig.* 459 D, E, F) evoke similar responses in the circular muscle from the cardia in that relaxation is the primary effect while eserinization reveals some contraction mediated through a cholinergic mechanism.

After cooling to 14° C. the more sensitive nerve elements in the preparation fail to respond to short-duration pulse-waves while the less sensitive muscle-fibres still respond to long-duration pulse-waves (*Fig.* 459 G).

Pattern of Innervation.—Having determined the direct response of muscle to adrenergic and cholinergic drugs, the pattern of innervation was studied by stimulation of the nervous elements in the œsophageal wall, using short-duration electrical pulses and nicotine. Although nicotine is classically held to stimulate only ganglion cells in the autonomic nervous system, a recent paper by Thompson (1958) suggests that it can also stimulate fine post-ganglionic nerve-fibres, and we have therefore considered a response to nicotine to indicate that some nervous structure, either ganglion cell or post-ganglionic fibres, is being stimulated.

Table IV.—The Pattern of Innervation of Muscle Strips on Electrical Stimulation

	Transverse Cardia	*Longitudinal Cardia*	*Transverse above Cardia*	*Longitudinal above Cardia*
0·03 m.sec., 200 volts	+ ↓	+ ↑	+ ↑	+ ↑
0·3 m.sec., 200 volts	+ ↑	+ ↑	+ ↑	+ ↑
Atropine 100 μg. 0·03 m.sec., 200 volts	+ ↓	O Block	O Block	O Block
Atropine 100 μg. 0·3 m.sec., 200 volts	+ ↑	+ ↑	+ ↑	+ ↑
Eserine 100 μg. 0·03 m.sec., 200 volts	+ ↓ ↑			
Atropine 100 μg. Eserine 100 μg. 0·03 m.sec., 200 volts	+ ↓			
Cool to 14° C. 0·03 m.sec., 200 volts	O			
Cool to 14° C. 0·3 m.sec., 200 volts	+ ↑			

+ ↑ = Responds by contracting; + ↓ = Responds by relaxing; + ↓ ↑ = Relaxation followed by contraction; O = No response.

DISCUSSION

In discussing these results it must be emphasized that we have been investigating the terminal portion of the autonomic nerves within the wall of the œsophagus and their relationship to the receptors in the œsophageal muscle. These studies give no indication of the anatomical pathways involved. Furthermore, we are only concerned with that portion of the human œsophagus below the arch of the aorta.

Response to Drugs.—Our findings indicate that there are two types of response to adrenergic drugs. Contraction is the usual response to adrenaline and noradrenaline, but, if these motor receptors are blocked by phentolamine, relaxation occurs. This dual response suggests that there are probably two types of receptors, one producing contraction and the other relaxation. These responses are much more marked at the cardia than elsewhere and are more marked in circular than in longitudinal muscle. In longitudinal muscle above the cardia there is no evidence of adrenergic receptors at all. From theoretical considerations Youmans (1952) suggested that a similar dual response may explain the movements of gastric muscle on autonomic stimulation.

In contrast, cholinergic drugs always produce contraction and there is no evidence of a cholinergic mechanism which will produce relaxation even after the motor elements have been blocked by atropine. That this contraction can always be completely abolished by atropine and is unaffected by succinyl choline indicates that below the aortic arch the human œsophagus behaves entirely as smooth muscle.

In the main body of the œsophagus nervous stimulation results in a contraction which can be augmented by eserine and blocked by atropine, and therefore must be mediated via cholinergic nerve-endings. The nerve-supply to the body of the œsophagus is therefore cholinergic.

At the cardia the position is more complicated. The circular muscle responds to nervous stimulation by relaxation, although there is a small element of contraction which can be augmented by eserine and blocked by atropine and is therefore cholinergic. The relaxation is similar to that produced by noradrenaline after phentolamine. It would seem reasonable therefore to suggest that some of the nerves supplying the cardia terminate around receptors which will produce relaxation when stimulated, and that the substance released at these nerve-endings is noradrenaline. It is possible, of course, that some other as yet unidentified substance is released which will also produce relaxation, but this seems an unnecessary multiplication of hypotheses. The remaining nerves terminate at cholinergic reaction and produce a contraction on stimulation.

In the longitudinal muscle at the cardia, although the innervation is predominantly cholinergic, there is occasionally some evidence of relaxation on nerve stimulation when the cholinergic element has been blocked.

The suggested innervation of the lower œsophagus and cardia may now be summarized and is represented diagrammatically in the figures indicated.

1. *The Lower Œsophagus.*—

a. Longitudinal muscle (*Fig.* 461): This muscle contains only cholinergic receptors. Nerve stimulation results in contraction mediated through these receptors.

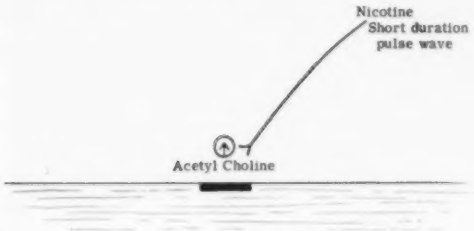

FIG. 461.—Pattern of innervation of longitudinal muscle above the cardia. ↑ Muscle responds by contracting.

b. Circular muscle (*Fig.* 462): While cholinergic receptors predominate the response to drugs indicates that there are a small number of adrenergic receptors. These are of two types, one producing contraction and one producing relaxation. Nerve stimulation causes the muscle to contract and acts through the cholinergic mechanism.

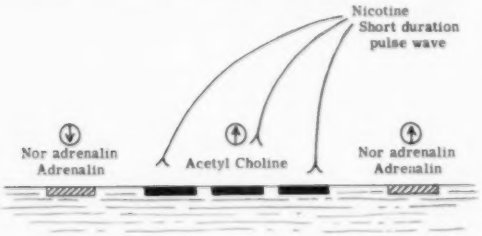

FIG. 462.—Pattern of innervation of circular muscle above the cardia. ↓ Muscle responds by relaxing; ↑ Muscle responds by contracting.

2. *The Cardia.*—

a. Longitudinal muscle (*Fig.* 463): Cholinergic and adrenergic receptors are present. In addition to producing contraction through the cholinergic receptors nerve stimulation occasionally results in relaxation. This is presumably mediated through the adrenergic relaxing mechanism.

b. Circular muscle (*Fig.* 464): Cholinergic and adrenergic receptors are present as in the preceding two groups of muscle strips. There is, however, a striking and almost constant difference in the response to nerve stimulation. Relaxation occurs and is achieved through the adrenergic receptors. Occasionally cholinergic receptors are stimulated and weak contraction results.

There are two interesting points which have emerged from this study. First, it has been shown without doubt that the circular muscle at the cardia of the human œsophagus has a pattern of innervation which differs from that of the rest of the lower œsophagus and thus provides some further evidence that the cardia is functionally a distinct entity.

Secondly, we have to some degree defined the parts played by the adrenergic and cholinergic nerves in the function of the lower œsophagus. These findings should, at least, suggest the lines on which drugs could be used systematically to modify the activity of this part of the œsophagus. Attempts have been made to treat achalasia of the cardia (Knight, 1934) by means of removal of various parts of the autonomic nervous system. Our results suggest that such attempts may be based on misapprehension as to the roles played by the sympathetic and parasympathetic systems and that further information is required as to the anatomical pathways involved before such surgery can be logically undertaken.

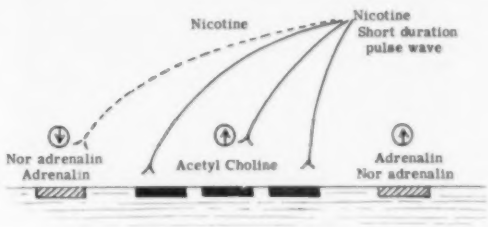

FIG. 463.—Pattern of innervation of longitudinal muscle at the cardia. Continuous line = main pathway. Dotted line = additional pathway; ↓ Muscle responds by relaxing. ↑ Muscle responds by contracting.

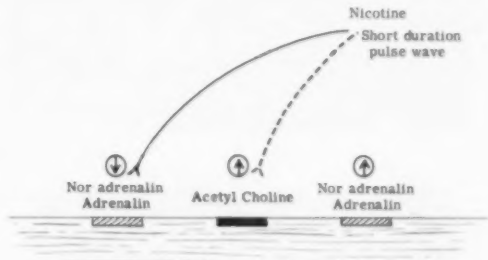

FIG. 464.—Pattern of innervation of circular muscle at the cardia. Continuous line = main pathway. Dotted line = additional pathway. ↓ Muscle responds by relaxing; ↑ Muscle responds by contracting.

SUMMARY

1. The response of isolated muscle strips from the lower part of the human œsophagus and the cardia to drugs and to electrical stimulation has been investigated.

2. The receptor mechanism of the muscle as far as its response to drugs is concerned, has been defined, and the pattern of autonomic innervation has been studied.

3. It has been concluded that the circular muscle of the cardia has a distinct pattern of innervation.

4. The significance of these findings has been discussed.

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EXCISION OF THE MAJOR DUCT SYSTEM FOR BENIGN DISEASE OF THE BREAST

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THE purpose of this paper is to describe a method by which the major duct system of the breast can be completely excised with preservation of the nipple and little or no reduction in the size of the gland. The operation, originally designed by Adair and Urban (personal communication) of the Memorial Center for Cancer, New York, has been practised by them and other American surgeons (Haagensen, 1951) over the last ten years, but it appears to be used far less frequently in this country. It is hoped that it will bridge the gap between local operations on a single duct and the more radical, and possibly less desirable, choice of a local mastectomy.

This report presents the results obtained in a series of 31 patients treated by excision of the major duct system of the breast for the following conditions:

1. Recurrent abscess of the major duct system which tends to point in the para-areolar region, to rupture in this situation and subsequently to form a chronic fistulous track communicating with one of the major ducts. This has been described by Atkins (1955) as *mammillary fistula*.

2. Bleeding from the nipple in patients with papillomatosis of the major duct system. The operation is of considerable value in those instances when bleeding from a single duct, easily demonstrable at the onset, cannot be found subsequently.

3. A recognizable subareolar cyst accompanied by a discharge from the nipple.

4. Persistent viscid whitish discharge from the nipple associated with generalized dilatation of the major duct below the nipple.

Table I gives a detailed analysis of the 31 patients treated by this method.

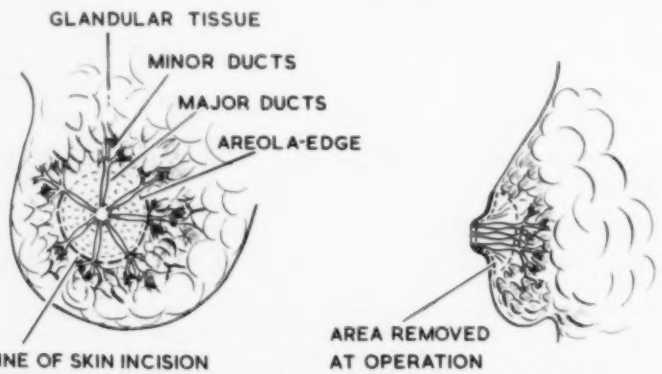
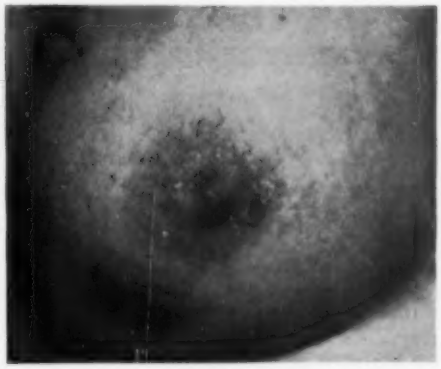


FIG. 465.—Diagram showing the anatomy of the major lactiferous ducts of the breast.

ANATOMY

Fifteen to twenty major or lactiferous ducts open separately at the nipple (*see Fig.* 465). From each arises a system of lobar ducts which are of three orders; the finest branches, i.e., those of the third

A



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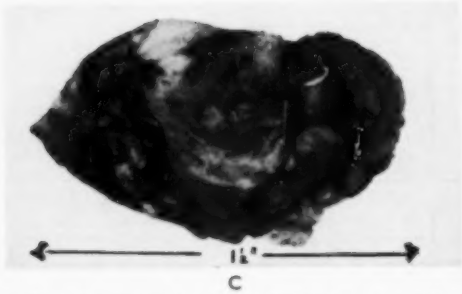

C

D

FIG. 466.—Results of operation (Patient No. 1, V. T.). A, Recurrent para-areolar abscess, May 23, 1956. B, Three weeks later: abscess has burst to form a mammillary fistula. C, One week later: operation for excision of major duct system of the breast. Specimen of excised duct system. D, Four weeks after operation—the final result.

order, give origin to the lobular ducts, the ultimate branches of which terminate in the glandular alveoli.

The major mammary ducts and the fat in which they lie form a truncated conical mass having a long axis of 1–1½ in., the diameter of its base being roughly that of the areola mammæ. It is this conical mass containing the major ducts which is removed.

OPERATIVE TECHNIQUE

The skin incision follows the lower half of the edge of the areola. If a sinus is present this is included in the skin incision and excised. The nipple is reflected from the breast tissue in the area covered by the areola and the lactiferous or major ducts divided as they enter the nipple skin. The mass containing the major ducts can then be removed as a single conical block of tissue either by excision with a knife and ligature of the bases of obvious major ducts with fine catgut or by cutting diathermy. The former takes a little longer but heals more satisfactorily. During dissection many dilated major ducts containing fluid can be identified.

The space remaining after excision is obliterated after hæmostasis has been secured by layers of fine catgut. The nipple, if originally inverted, is gently everted and tacked down to the breast substance with a fine catgut stitch. In order to keep the nipple everted a purse-string suture of catgut may be used, but is not always necessary. The skin is closed with fine silk or nylon and a small strip of corrugated rubber to drain the subcutaneous tissues is brought through the incision. This is removed after 24 hours by pulling on a long thread stitch which is attached to its end, brought out through the centre of the dressing, and secured on the outside with a piece of strapping. The wound is dressed with gauze and wool. While the wound is healing every effort should be made to avoid tension on

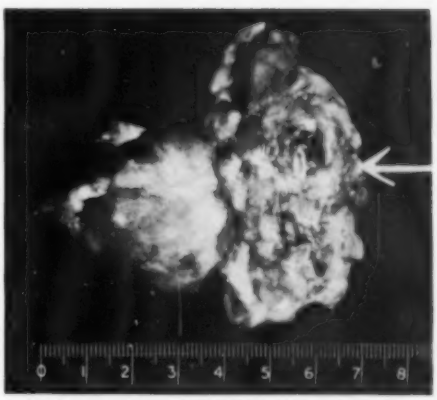

FIG. 467.—Specimen of major duct system and retention cyst removed from Patient No. 20 (E. J.) at operation. The arrow indicates the site of the nipple. The major ducts were excised with the cyst on account of its size and the associated symptoms.

Table I.—ANALYSIS OF A PERSONAL SERIES OF 31 CASES TREATED BY EXCISION OF THE DUCT SYSTEM OF THE BREAST

PATIENT	AGE	PRESENTING SYMPTOM	LENGTH OF HISTORY (WEEKS)	SIDE	NIPPLE	LACTATION	LENGTH OF FOLLOW-UP (MONTHS)	PATHOLOGICAL FINDINGS
Recurrent Para-areolar Abscess and Mammillary Fistula:—								
1. V. T.	32	Para-areolar abscess	12	R.	Retracted	Never pregnant	24	
2. T. N.	29	Mammillary fistula	6	R.	Retracted	Never pregnant	18	
3. M. G.	33	Para-areolar abscess	1	R.	Retracted	Abnormal due to nipple	24	
4. A. B.	34	Mammillary fistula	16	L.	Retracted	Abnormal due to nipple	12	The general picture in this group was constant—a fistulous track passing down and communicating with one duct of the major duct system, the wall of which was lined by squamous epithelium. The major ducts were generally dilated and many of them contained amorphous debris
5. J. T.	28	Mammillary fistula	8	L.	Retracted	Never pregnant	12	
6. M. M.	30	Para-areolar abscess	1	R.	Normal	Never pregnant	36	
7. W. S.	29	Para-areolar abscess	2	L.	Retracted	Abnormal due to nipple	36	
8. E. M.	27	Para-areolar abscess	1	R.	Retracted	Never pregnant	30	
9. A. N.	35	Mammillary fistula	20	R.	Retracted	Never pregnant	24	
10. M. M.	31	Para-areolar abscess	1	R.	Retracted	Never pregnant	18	
Duct Papilloma of the Breast:—								
11. A. S.	64	Bleeding from nipple	1	L.	Normal	Yes: normal	18	Generalized dilatation of duct system: multiple papillomata throughout main ducts. 6 yr. ago—excision of single duct in same breast on account of bleeding
12. L. H.	64	Bleeding from nipple	3	L.	Normal	Yes: normal	12	Generalized duct dilatation with papilloma confined to single bleeding duct
13. F. B.	68	Bleeding from nipple	2	R.	Normal	Yes: normal	9	Papilloma in one duct. Other major ducts—slightly dilated
14. B. Z.	71	Bleeding from, and lump above, nipple	3	L.	Normal	Never pregnant	15	11 yr. ago—excision of single duct for papilloma; on same side section now shows many small papillomata in major ducts
15. M. C.	48	Bleeding from, and lump above, nipple	1	R.	Normal	Never pregnant	6	Single papilloma in one duct: others appear normal
16. R. C.	45	Bleeding from nipple. Persistent green discharge from nipple	2 24	L.	Retracted	Never pregnant	12	Simple papilloma in one major duct: rest dilated and contain greenish fluid
17. F. E. W.	49	Thick whitish discharge from nipple: recently bloodstained	28 1	L.	Normal	Yes: Normal	5	Simple papilloma in one major duct: rest dilated and contain whitish fluid
18. E. N. G.	39	Tender, para-areolar swelling: bloodstained discharge from nipple. Whitish discharge from nipple	3 30	R.	Normal	Never pregnant	9	Simple papilloma in one major duct: rest dilated and contain whitish fluid
19. M. A.	41	Firm tender swelling in sub-areolar area and viscid white discharge. Bleeding from nipple	12 2	R.	Retracted	Abnormal due to nipple	5	Simple papilloma in one major duct: others dilated and filled with secretion in area of subacute mastitis
Retention Cyst of the Duct System:—								
20. E. J.	49	Painful swelling behind nipple	16	L.	Normal	Yes: Normal	20	Cyst = 5 × 4 cm. Contained mobile green fluid
21. E. G.	40	Clear discharge from nipple: tenderness	24	R.	Normal	Never pregnant	4	Cyst = 3 × 2 cm. Contained opalescent fluid
22. J. A.	41	Para-areolar swelling	44	L.	Normal	Never pregnant	3	Cyst = 1 cm. diameter. Contained colourless fluid In this group there was generalized dilatation of the whole major duct system with the formation of a central, thin-walled cyst lined by flattened cubical epithelium
Chronic Discharge from the Nipple:—								
23. A. C.	47	Para-areolar swelling and white discharge	104	L.	L. inverted; R. n.a.d.	Abnormal due to nipple	30	Dilated ducts lined by squamous epithelium in an area of subacute mastitis
24. L. S.	44	Green discharge from nipple	130	R.	Both inverted	Never pregnant	18	

Table I.—Analysis of a Personal Series of 31 Cases treated by Excision of the Duct System of the Breast (*continued*)

Patient	Age	Presenting Symptom	Length of History (Weeks)	Side	Nipple	Lactation	Length of Follow-up (Months)	Pathological Findings
Chronic Discharge from the Nipple (*cont.*):—								
25. M. M.	37	Thick white discharge: crusting of nipple	78	R.	Both inverted	Never pregnant	12	Dilated ducts in mature fibrous tissue
26. J. W.	39	Thick white discharge: tender breast	156	R. L.	Both inverted	Never pregnant	48	
27. B. E.	48	Colourless discharge: heavy feeling in breast	208	L.	Normal	Yes: Normal	24	Dilated ducts filled with secretion. Generalized flattening of epithelium with two areas showing epithelial hyperplasia in the ducts
28. A. W.	50	Whitish discharge from nipple: tender breast	156	R. L.	Normal	Yes: Normal	12	
29. C. H.	52	Whitish discharge from nipple: tender breast	208	R. L.	Both inverted	Abnormal due to nipple	6	Dilated ducts filled with secretion. Generalized flattening of duct epithelium. In one area of three major ducts epithelium has become squamous in type
30. E. W.	55	Recent thick whitish discharge from right nipple. Small amount colourless discharge from left nipple	120	R.	R. inverted L. normal	Abnormal due to nipple	3	Dilated ducts filled with secretion. Generalized flattening of epithelium in an area of subacute mastitis
31. R. W.	43	Thick greenish discharge from left nipple	52	L.	Both normal	Normal: 2 children	5	Dilated ducts filled with secretion. Generalized flattening of the epithelium with occasional areas of epithelial proliferation

the stitches which obliterate the cavity below the nipple. Any dressing which adequately lifts the breast up from below will do this, elastoplast being suitable. For patients sensitive to elastoplast good support can be obtained by using a newly laundered brassière, which provides a more satisfactory, less cumbersome, support than any form of bandage (*Figs.* 466, 467).

PATHOLOGY

An early and probably the first description of this condition was published in 1850 by John Birkett, surgeon to Guy's Hospital, who became President of the Royal College of Surgeons of England. In his account of patients with retention cysts of the major duct system and chronic discharge from the nipple, he stated that the basic lesion was generalized dilatation of the major ducts due to obstruction at the nipple either by an inverted nipple or by plugging of the duct mouths with inspissated secretion. He believed that the end-result was cyst formation originating in blocked dilated ducts with atrophy and flattening of their lining epithelium. He made no mention of para-areolar abscess or chronic sinus formation. The true nature of these conditions was first described by Zuska, Crile, and Ayres (1951) as 'mammary duct fistula'. Its relation to recurrent para-areolar abscess, suggested by Kilgore and Fleming in 1952, was fully confirmed and the term 'mammillary fistula' was used by Atkins in 1955.

Observations in this series confirm the finding of other authors in that patients with mammillary fistula invariably have some associated nipple deformity which gives rise to inadequate drainage of the duct system of the breast externally. This view is supported by the experience that when the disease is unilateral it is found on that side which has an abnormal nipple, the gland having a normal nipple remaining unaffected.

In patients who benefit from excision of the major breast ducts, deformity of the nipple though often present is not invariable. In one group, characterized by persistent discharge from a nipple which may show no deformity, duct obstruction was found to be related to plugging of a main duct by inspissated secretion. Many of the patients in this group were post-menopausal and the duct stasis may be related

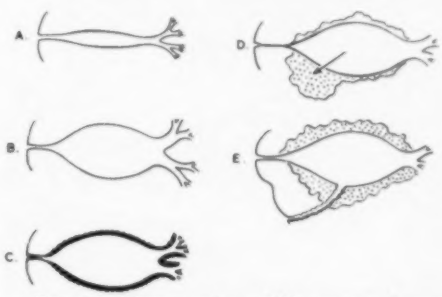

Fig. 468.—Diagram tracing possible sequence of events after major duct obstruction. A, Normal duct. B, Dilated duct after obstruction at nipple. Total obstruction will give rise to a retention cyst of the major duct system at this stage. C, Dilated duct with subacute periductal inflammation. Partial obstruction in B and C will give rise to a chronic discharge from the nipple. D, Dilated duct totally obstructed. Contents of the lumen are secondarily infected—may burst to produce para-areolar abscess. E, Mammillary fistula formation. Fistula opens into a major duct from the skin at the areolar margin.

to lack of the normal secretion by a breast no longer subject to hormone stimulation; a possible sequence of events is suggested in the accompanying diagram (*Fig.* 468).

Atkins (1955) and, later, Patey and Thackray (1958) have shown that the mammillary sinus connects directly with a single major duct to form a fistula. In the series here reported this finding has been repeatedly confirmed at operation. Our observations differ from those of Patey and Thackray with

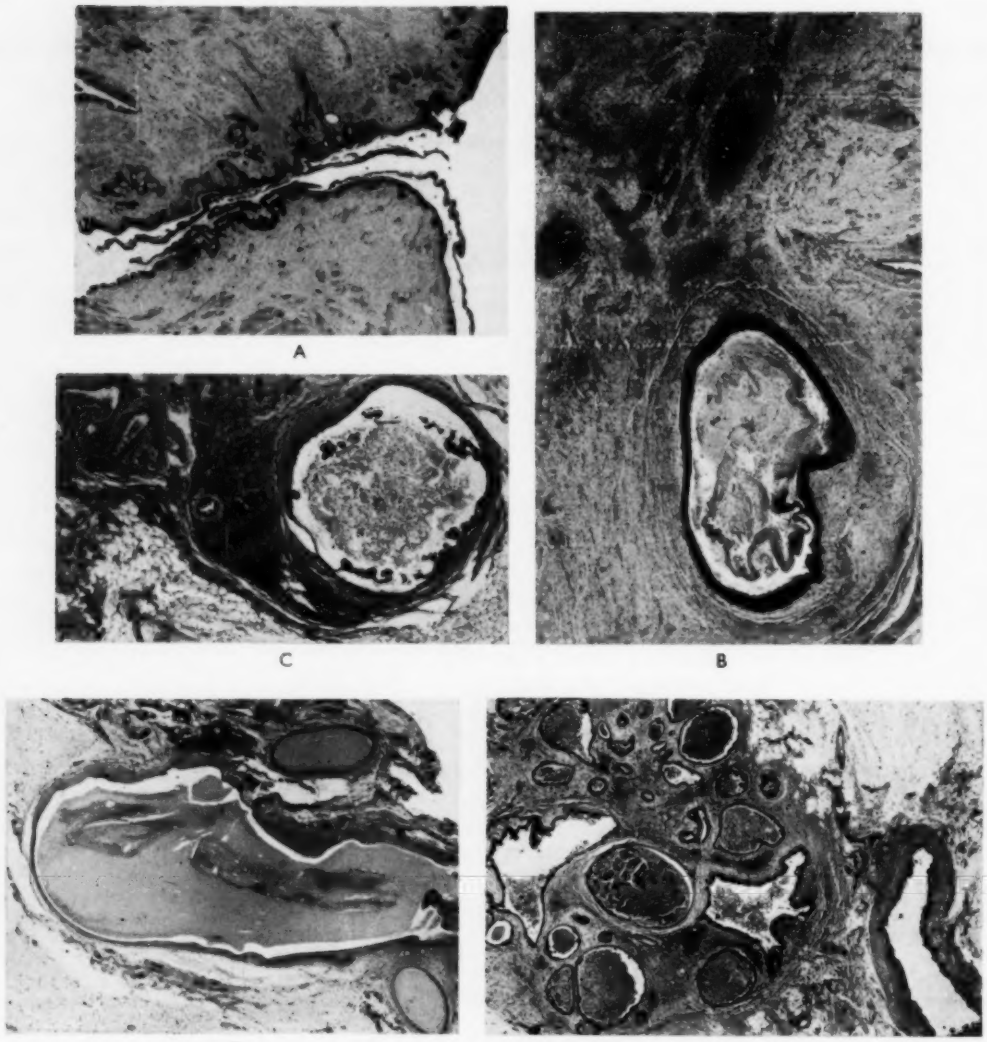

FIG. 469.—A, Mammillary fistula between the skin and the major duct lined by squamous epithelium. (× 7·5.) B, Main duct lined by metaplastic squamous epithelium and obstructed by amorphous debris and desquamated squamous epithelium. Note the diffuse focus of densely packed, subacute inflammatory cells—the majority are mononuclear. There are several collections of foreign-body giant cells. (× 15.) C, An obstructed duct containing fatty debris and showing a minor degree of epithelial proliferation and a focus of subacute inflammation. (× 15.) D, A main duct showing general dilatation with considerable atrophy of the lining epithelium. Minor inflammatory changes are present. (× 7·5.) E, A group of occluded branching ducts obstructed by fatty debris, with a minor degree of surrounding subacute inflammation. (× 15.)

regard to the situation of the junction between the fistulous track and the major duct. In our experience the track passes medially from its ostium at the areolar margin to join the major duct at the proximal part of the ampulla.

Histological study of serial sections of operation specimens of mammillary fistula shows that many, if not all, of the major ducts are affected in a varying degree (*Fig.* 469): Squamous metaplasia in the sinus and its duct, general distension of the affected duct, subacute to chronic inflammation and the production of a granulomatous mass in the interstitial tissue around the fistula and its duct, and diffuse spreading of the granulomatous region to involve a large part of the interstitial tissue in which the major ducts lie.

SUMMARY AND GENERAL COMMENTS

A method is described by which the whole major duct system of the breast can be removed.

The pathological picture of generalized dilatation of the major duct system with chronic discharge from the nipple, para-areolar abscess, and mammillary fistula and retention cysts of the major duct system is described. It is suggested that the basic lesion common to all three conditions is major duct obstruction followed by dilatation of tributary branches below the obstruction.

It is suggested that this operation may be useful in the treatment of patients with non-malignant disease of the major ducts. In the treatment of mammillary fistula in younger patients sacrifice of breast function is a serious disadvantage, but it is doubtful whether these glands, many of which have an inverted nipple, ever reach the stage of satisfactory functional competence. The alternative would be the simple operation of laying the fistulous track open, as described by Atkins (1955). It may be that the operation here described should be reserved for those patients in whom the condition recurs after local operation, and for the very severe cases. Our own observations, and those of Adair and Urban, confirm that the operation makes no difference to breast size. None of our patients has become pregnant since operation, but Adair and Urban have found that when young women who have had the major duct system excised on one side became pregnant, normal lactation will take place in the intact breast provided that its nipple is not inverted. Furthermore, if pregnancy supervenes within a year of operation there may be no discomfort, apart from some slight tenderness, in the breast, the major ducts of which have been excised. After this period there is sufficient atrophy of the glandular tissue of the breast for pregnancy to have little or no effect.

In our experience there have been no untoward effects on the mammary glands of women in whom the whole duct system has been excised. There is little or no information regarding the rate of recurrence after excision of the fistulous track in large numbers of cases after a relatively long post-operative period. In the absence of this information it would seem rational to excise the whole diseased area for a condition which is so often generalized. In patients with chronic nipple discharge in whom the disease often affects more than one duct, and many are over the age of 45 years, major duct excision holds out the best prospect for cure.

This operation has been used in duct papilloma of the breast occurring in oldish women in whom sacrifice of the major duct system presents no problem. Localization and excision of the single bleeding duct in such patients is often satisfactory and adequate, but the operation described in this paper is very little longer or more extensive and it eliminates the possibility of further recurrence. In *Table I* it will be seen that Patients 11 and 14 had had a previous local operation for excision of a single duct papilloma; they reported with recurrence six and eleven years respectively after the first local excision. In the series of patients in whom the major duct system has been excised for a clinically single papilloma, multiple papillomata not suspected on clinical examination have not infrequently been found in other ducts after careful dissection of the operation specimen.

A simple retention cyst of the duct system often occurs as a tiny subcutaneous swelling at the junction of the skin and areola in a young woman with a normal nipple. Under these circumstances excision of the whole major duct system is not indicated as the condition can be adequately and satisfactorily treated by a local excision of the cyst.

Acknowledgements.—I would like to thank Drs. Adair and Urban, of the Memorial Hospital, New York, who taught me the technique of the operation; and Professor Sir James Paterson Ross for his advice and guidance. I am indebted to Mr. E. V. Willmott for the photomicrographs.

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DUPLICATIONS OF THE ALIMENTARY TRACT

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THE usual definition of duplication of the alimentary tract is a spherical or tubular structure closely applied to some part of the digestive tract. Meckel's diverticulum, which is covered by this definition, is specifically excluded. In our experience the words 'closely applied' are not always justified, as we have encountered duplication cysts in the mediastinum separated by more than 1 in. from the œsophagus. According to Ladd and Gross (1940) an alimentary duplication is lined by mucosa which could have been derived from endoderm. Such names as enterogenous cyst, giant diverticulum, ileum duplex, etc., are confusing. We propose to adhere to the inclusive term 'duplication'.

During the last eight years 28 children with alimentary duplications have been operated upon in the two Liverpool Children's Hospitals. The population of the area is about a million and practically all children are admitted to these two hospitals. It would not, however, be justifiable to calculate the frequency of duplication from these figures, as many children are also drawn from a wider area. Instances of duplication found at autopsy are not included.

PATHOGENESIS

Several theories of the origin of alimentary tract duplications have been postulated. The phylogenetic explanation of Hudson (1935) is based on the double cæca of fowls.

Lewis and Thyng (1907) observed clumps of epithelial cells which formed pouches related to the œsophagus, stomach, and intestine in pig and rabbit

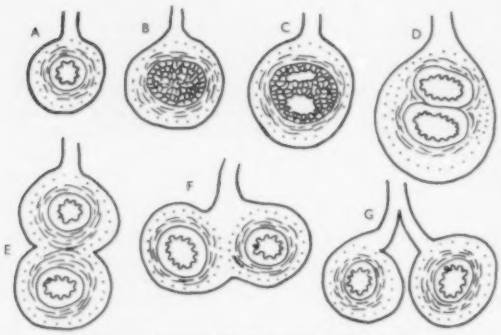

FIG. 470.—Diagram of development of duplication. **A**, Early stage of alimentary tube; **B**, Solid stage; **C**, Two vacuoles in cell mass; **D**, Two epithelial lumina with conjoined basement membranes; **E**, Conjoined circular muscle layers between hollow structures; **F**, Same in horizontal position which can progress to— **G**, Free duplicate tubes, each with complete intestinal wall and separate mesentery.

embryos of between the fourth and eighth week of intra-uterine life. These epithelial cell-nests usually disappear, but may occasionally persist, become separated from the alimentary tract lumen, and by continued growth develop into duplications. This theory is not entirely acceptable: the epithelial pouches of pig and rabbit embryos usually occur along the antimesenteric border of the alimentary tract, duplications being almost invariably located along the mesenteric aspect of the digestive tube.

Bremer (1944) divides duplications into spherical and tubular forms. He states that during the sixth to seventh week of intra-uterine life the intestine goes through a "solid stage" during which the lumen is obliterated by an accumulation of epithelial cells. Later, vacuoles appear in this solid cell mass and coalesce to form longitudinal rows parallel to the long axis of the gut. The walls of these rows of vacuoles finally break down to establish the definitive bowel lumen. Bremer considers that spherical duplications are the result of abnormal persistence of vacuoles. He states that tubular forms are derived from true diverticula, which are not infrequently present along the mesenteric border of the alimentary canal of the human embryo, between the eighth and ninth week of intra-uterine life. We have not encountered a case like that depicted in *Fig.* 470 G.

Ravitch (1953), in his masterly article on hind-gut duplications, states that doubling of the colon and genito-urinary apparatus and varying degrees of doubling of the lumbosacral spine can be explained as a form of partial twinning or as incomplete fusion of the two individuals. Fusion of two early amphibian embryos has been observed to occur. Ravitch believes that caudal doubling probably occurs during the first two weeks of embryonic life.

Holt and McIntosh (1953) suggest that duplication is the result of midline splitting of primordial tissue. Black and Benjamin (1936) postulate that duplications in the chest result from intrathoracic remnants of the omphalomesenteric duct.

The most commonly accepted hypothesis is the notochordal theory of Veeneklass (1952) for mediastinal lesions. This theory explains the fact that in cases of mediastinal duplication there is usually an associated vertebral anomaly. The notochord first appears during the third week of embryonic life, its 'head process' wedging itself between the cells forming the endoderm (*Fig.* 471). Should there be incomplete separation of the notochord from the endoderm, traction may be exerted upon the primitive gut in such a way that a pouch of gut is drawn towards the vertebral bodies. Loss of continuity between the pouch and the primitive gut from which it arose combined with continued growth of the pouch, results in a duplication (*Fig.* 471).

The incidence of duplication in different positions of the alimentary tract in this series was as follows:—

Site	*Number*	*Percentage*
Buccal or pharyngeal	0	0
Mediastinum (only)	4	14
Stomach	1	4
Duodenum	3	11
Jejunum	1	4
Ileum and ileocæcal angle	11	39
Small intestine and mediastinum	2	7
Cæcum	3	11
Colon	1	4
Rectum	2	7

The ileum in our cases was, therefore, the commonest site of duplications; this is comparable to

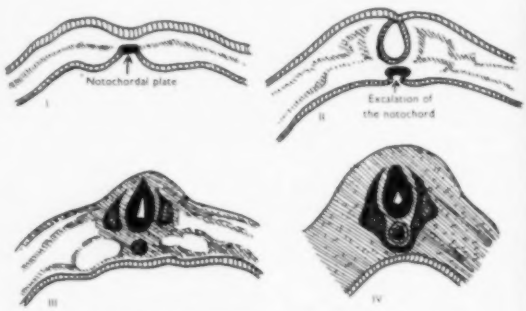

FIG. 471.—Showing excalation of the notochord and its subsequent envelopment by the developing spinal column. (After Arey, Brash, and Keith.)

the findings of other authors. Dohn and Povlsen (1951) reviewed the literature and found that about 50 per cent of published instances of duplication occurred in the ileum; 40 per cent of Gross's (Gross, Holcomb, and Farber, 1952) series were ileal.

PATHOLOGY

Although we have seen cysts in the tongue lined by squamous or flattened epithelium, we have not encountered a definite alimentary duplication cyst

situated in the tongue. The only record of an alimentary duplication cyst situated in the tongue was lined by colonic epithelium (Gross and others, 1952). This duplication must be buccal or pharyngeal, though its situation may be lingual.

External Appearance.—Duplications may be:

1. Spherical, cystic structures filled with clear mucoid fluid (22 cases in our series).

2. Tubular structures extending along a variable length of gut between the leaves of the mesentery,

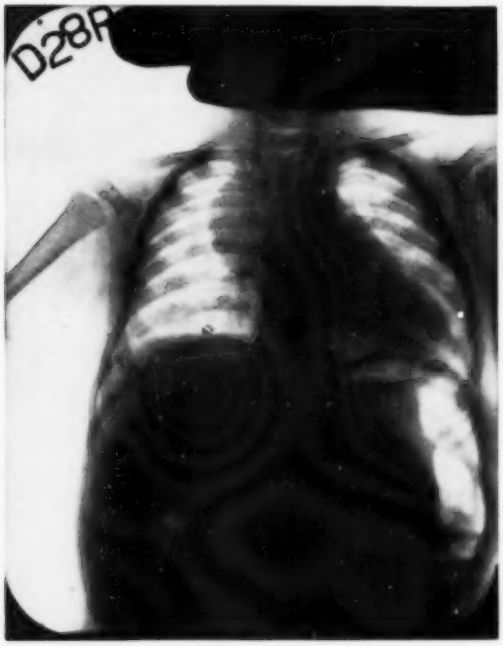

FIG. 472.—*Case* 18. Radiograph showing mediastinal cyst.

the duplication communicating by one or both extremities with the normal intestinal channel (5 cases in our series). The duplication may also be blind at both ends.

3. Double-barrelled segments of intestine (3 cases in our series).

4. Simulating a new growth situated externally (1 case in our series). There was one very long and bizarre structure amongst our cases (*Case* 2). Although the gut was gangrenous at operation, it was possible to make out that over a considerable distance there were three intestinal tubes running parallel to each other; this, therefore, was really a 'triplication' of the small bowel, which appears to be very rare. We have been able to find only one reference in the literature—Gray (1940) described a case of colonic triplication.

Mucosal Lining.—In 14 of our cases the duplication was lined by gastric epithelium. Peptic ulceration may occur in this ectopic mucosa.

Multiple Duplications.—More than one duplication may occur either in the abdomen or chest or in both. In this series there were 2 cases with two intestinal duplications (*Cases* 19 and 27); 1 case with one duplication in the abdomen and one in the chest (*Case* 7); 1 case with a tubular duplication in the abdomen and two duplication cysts in the mediastinum, one on either side of the spine (*Fig.* 472, (*Case* 18). Roth (1881) was the first to report a case in which an abdominal and mediastinal duplication were present in the same patient, and since then there have been several reports in the literature of this combination (Johnston, Hallenbeck, Ochsner, and Du Shane, 1954; Fallon, Gordon, and Lendrum, 1954). In *Case* 18 a tubular jejunal duplication thinned out to a cord-like structure and ran upwards behind the duodenum to penetrate the diaphragm. Duplications arising from the intestine and penetrating the diaphragm are very uncommon: a few having been reported in the literature (Gross, Neuhauser, and Longino, 1950). In *Case* 12 a thick cord-like structure descended from a mediastinal cyst, but tapered off to a thin thread at the level of the diaphragm.

Associated Malformations.—Most observers agree that duplication is often associated with other malformations. This series is no exception, as can be seen from *Table I*. In *Cases* 1 and 3 associated malformations were so many and so severe as to cause the child's death.

As has previously been mentioned, vertebral anomalies may be associated with intrathoracic duplications. Fallon and others (1954) reported 18 cases with spina bifida anterior, hemivertebra, or infantile scoliosis in 61 cases with mediastinal duplication cysts. Ware and Conrad (1953) reviewed the literature and found that 18 out of 81 cases with intrathoracic cysts had spinal abnormalities. Veeneklaas (1952) stressed the importance of tomography in discovering vertebral defects. Gross and others (1950) mentioned a case with intrathoracic duplication cyst associated with Klippel-Feil syndrome, and one of Ladd and Scott's (1941) cases had brevis collis. There is no record of a vertebral anomaly associated with a purely intra-abdominal duplication. Two of our four cases of intrathoracic duplication without an abdominal duplication had a dorsal spinal abnormality. It is likely that as this combination of malformations is now widely recognized, more instances will be observed in the future.

CLINICAL FEATURES

1. Intrathoracic Lesions.—Mediastinal duplication cysts were first described by Von Wyss in 1870. They are commoner on the right than on the left side in the ratio of 5 : 1 (Olenik and Tandatnick, 1946). Mediastinal duplications occur in either sexes with about equal frequency. These cysts always occur in the posterior mediastinum and are usually closely applied to a vertebra. They are stated to be closely applied to the wall of the œsophagus, but this is not always the case. In *Case* 18, which had bilateral mediastinal cysts, both cysts were situated at a distance from the œsophagus.

A mediastinal duplication cyst may be completely silent (*Case* 16) and discovered only on routine radiological examination or at autopsy. A large cyst or a small cyst in certain situations will exert pressure upon vital structures, and cause dyspnœa,

Table I.—DETAILS OF 28 CASES OF DUPLICATIONS OF THE ALIMENTARY TRACT

CASE NO.	AGE	SEX	PATHOLOGY	ASSOCIATED MALFORMATIONS	PRESENTING SIGNS AND SYMPTOMS	TYPE OF OPERATION	COMPLICATIONS	RESULT
1	1 d.	M.	Double ascending colon ending blindly at both ends	Vesico-intestinal fissure with diphallus, imperforate anus, pelvic kidneys, sacral spina bifida, exomphalos, and ectopia vesicæ		Resection of colon and construction of perineal ileostomy		Died 2 mth. later from pyelonephritis
2	1½ d.	M.	Tubular triplication of ileum, volvulus gangrene and perforation. Meconium peritonitis		Intestinal obstruction, shock	Resection of over ¾ of small intestine		Died
3	2 d.	M.	Duplication cyst with gastric and pancreatic mucosa in posterior mediastinum	Œsophageal atresia with fistula, absence of right lung, and a two-chamber heart	Cyanosis	Excision of cyst and repair of œsophageal atresia	Cardiac failure and sclerema	Died
4	3 d.	M.	Cystic duplication (7 × 6 × 3 cm.) second and third part duodenum	Talipes	Intestinal obstruction	Excision of duplication and adjoining duodenum. Duodenostomy	Obstructive jaundice due to scarring cholecystoduodenostomy	Recovered
5	4 d.	M.	Duplication cyst (2 × 1·5 × 1 cm.) of cæcum lined by intestinal mucosa causing ileocolic intussusception		Vomiting, blood P.R.	Reduction of intussusception and enucleation of cyst		Recovered
6	5 d.	M.	Cyst of cæcum lined by colonic mucosa		Vomiting, abdominal distension	Excision of cyst, adjacent ileum, and cæcum. Ileocolostomy		Recovered
7	8 d.	F.	(1) Tubular ileal duplication lined by gastric, pancreatic, and ileal mucosa, perforation, peritonitis. (2) Mediastinal cyst lined with mucus-secreting columnar cells		Symptoms and signs of advanced peritonitis	Resection of duplication and adjacent ileum. End-to-end anastomosis		Died of peritonitis
8	8 d.	F.	Duplication cyst of rectum (2·5 × 1 cm.) containing a linear cavity communicating with introitus of vulva	Right pelvic kidney on I.V.P.	Bizarre lump in the vulva mainly below the vaginal opening	Excision of duplication along with part of the labium majus. Careful dissection of the vagina and rectum		Recovered
9	10 d.	M.	Duplication cyst (10 × 6 × 6 cm.) of ileocæcal junction lined by intestinal mucosa	Hallux primus rigidus	Vomiting and abdominal mass	Resection of cyst and adjacent ileum, cæcum, and ascending colon. Side-to-side ileocolostomy		Recovered
10	10 d.	F.	Tubular duplication of ileum (60 cm. long)	Malrotation of midgut	Vomiting and abdominal mass	Resection of duplication and adjoining ileum. End-to-end anastomosis		Recovered
11	4 wk.	F.	Duplication cyst (9 × 6 × 6 cm.) of upper jejunum lined by gastric mucosa		Signs of intestinal obstruction	Excision of cyst and adjacent jejunum. End-to-end anastomosis	Re-obstructed 3 mth. later owing to peritoneal bands which were divided at second laparotomy	Recovered
12	5 wk.	M.	Duplication cyst of terminal ileum, lined by gastric mucosa, volvulus and gangrene of ileum, ascending and transverse colon	(1) Myelomeningocele (2) Meckel's diverticulum	Shock, abdominal mass and vomiting	Resection of cyst and gangrenous gut. End-to-end ileocolostomy		Died
13	5 wk.	M.	Duplication cyst posterior mediastinum (2·5 cm. diam.) lined by intestinal mucosa. Calcification in wall of cyst	Abnormality of middorsal spine. Congenital hypertrophic pyloric stenosis	Cyanosis	(1) Excision of cyst. (2) Rammstedt's operation		Recovered
14	6 wk.	F.	Duplication cyst of pylorus containing hæmorrhagic mucosa		Projectile vomiting, palpable pyloric tumour	Excision of cyst and a wedge of pylorus		Recovered

Case No.	Age	Sex	Pathology	Associated Malformations	Presenting Signs and Symptoms	Type of Operation	Complications	Result
15	2 mth. (*Fig.* 476)	M.	Duplication cyst (3 × 4 × 4 cm.) of cæcum lined by columnar mucus-secreting cells. Gastric mucosa without oxyntic cells. Respiratory epithelium with mucous glands of bronchial type		Signs of intestinal obstruction and mass in R.I.F.	Resection of cyst and adjacent ileum, cæcum, and ascending colon. End-to-end anastomosis		Recovered
16	3 mth.	M.	Duplication cyst posterior mediastinum (3 × 2 cm.) lined by tall columnar epithelium	Cervical meningocele separation of pedicle upper dorsal vertebræ	Cyst found at routine X-ray examination of chest	Excision of cyst (the meningocele to be removed later)		Recovered
17	3 mth.	M.	Duplication cyst (1 cm. in diameter) of ileum lined with gastric mucosa at apex of ileo-ileal intussusception which was gangrenous		Vomiting, abdominal mass blood-stained mucus P.R.	Resection of intussusception. End-to-end anastomosis		Recovered
18	4 mth.	M.	(1) Tubular duplication (20 cm. long) of jejunum lined by gastric mucosa, perforation, peritonitis. (2) Duplication cyst in left posterior mediastinum. (3) Duplication cyst in right posterior mediastinum	Hemivertebra, D.3 and 4	Signs of peritonitis and blood P.R.	(1) Resection of duplication, adjacent jejunum, and upper ileum. (2) Fifteen days later excision of right mediastinal cyst. (3) Eight months later excision of left mediastinal cyst		Recovered
19	6 mth.	M.	(1) Duplication cyst of ileum removed when 9 wk. old. (2) Tubular duplication of ileum (20 cm. long) lined by gastric and ileal mucosa		Blood P.R. Marked anæmia necessitating repeated blood transfusions	Resection of duplication and adjoining ileum. End-to-end anastomosis		Recovered
20	6 mth.	F.	Duplication cyst in posterior mediastinum lined by gastric mucosa			Excision of cyst	Partial collapse left lower lobe	Recovered
21	16 mth.	M.	Duplication cyst (4 × 2·5 × 2·5 cm.) of ileocæcal junction lined by gastric ileal and colonic mucosa		Vomiting and abdominal mass	Enucleation of cyst		Recovered
22	4 yr.	M.	Duplication cyst of duodenum lined by gastric mucosa. Perforation causing peritonitis		Signs of advanced peritonitis and shock. Blood P.R.	Resection of cyst and adjacent duodenum. Duodenoduodenostomy	Hypostatic pneumonia	Died
23	4 yr.	M.	Duplication cyst (4 cm. in diam.) of ileocæcal junction lined by intestinal mucosa		Abdominal pain and vomiting	Resection of cyst, adjacent ileum, and part of cæcum. End-to-side anastomosis		Recovered
24	5 yr. (*Fig.* 477)	M.	Tubular duplication (50 cm. long) of ileum lined by gastric mucosa. Perforation with generalized peritonitis		Signs of advanced peritonitis	Resection of duplication and adjacent ileum. Double-barrelled ileostomy. Drainage of peritoneal cavity		Died
25	6 yr.	M.	Duplication cyst of rectum lined by columnar epithelium		Rectal prolapse	Excision of cyst only		Recovered
26	7 yr.	F.	Duplication cyst (2 cm. diam.) of ileocæcal angle lined by columnar epithelium		Vomiting, pain in R.I.F.	Resection of cyst and adjacent ileum and cæcum. Side-to-side anastomosis		Recovered
27	10 yr.	F.	(1) Duplication cyst (14 × 6 × 5 cm.) of ileum lined by gastric mucosa. Eight acute peptic ulcers. (2) Tubular duplication (25 cm. long) lined by ileal mucosa		Abdominal pain and vomiting, abdominal mass	Resection of cyst and tubular duplication with adjoining ileum. End-to-end anastomosis		Recovered
28	13 yr.	F.	Duplication cyst (2 × 3 × 4 cm.) of duodenum lined by gastric mucosa. Ulceration into common bile-duct, with stone in latter	Double gall-bladder	Obstructive jaundice, pain in the right hypochondrium	Resection of cyst and adjacent duodenum. Duodenoduodenostomy. Removal of stone and drainage of common duct. Cholecystectomy		Recovered

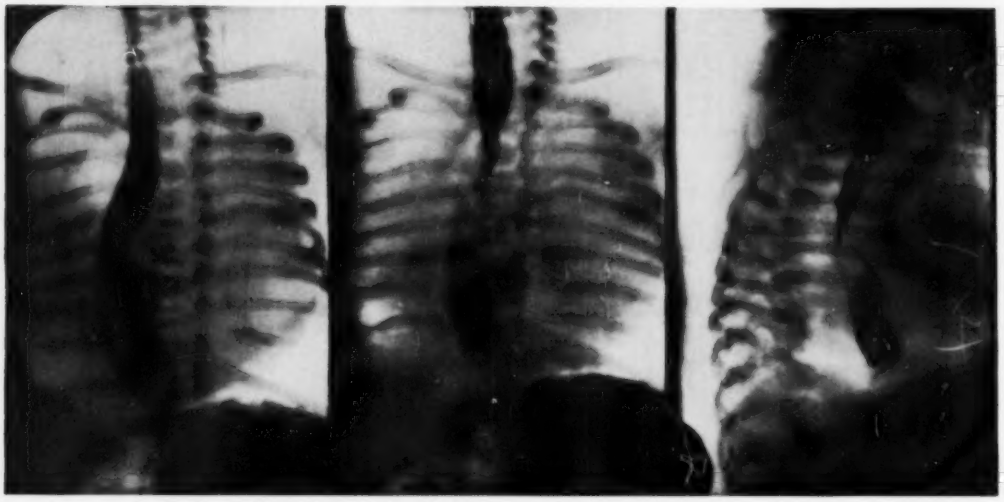

FIG. 473.—*Case* 13. Barium showing displacement of œsophagus due to mediastinal cyst.

FIG. 474.—*Case* 8. Duplication of rectum presenting at the vulva.

FIG. 475.—*Case* 8. Post-operative photograph of *Fig.* 474.

cyanosis, or dysphagia in this order of frequency, or a combination of these symptoms may result. Clinical examination may reveal an area of dullness due to the cyst itself or to pulmonary collapse from pressure on lung, bronchus, or trachea. Signs of cardiac displacement may be apparent. Pressure on or displacement of the œsophagus or trachea may be demonstrated radiologically after barium swallow or lipiodol injection (*Fig.* 473, *Case* 13).

Complications.—A cyst lined with gastric mucosa is liable to peptic ulceration leading to perforation; the symptoms will depend upon the situation of the cyst and the anatomical relationships of the point at which perforation occurs. There may be mediastinitis

or if the lung tissue is eroded, hæmoptysis, lung abscess, empyæma, or pyopneumothorax.

Bickford (1949) reported 40 cases with mediastinal cysts, 24 of which were lined by gastric mucosa; of our 3 cases 2 had gastric mucosa in the cyst lining—neither had any of the above complications.

2. Intra-abdominal Lesions.—It is well recognized that intra-abdominal duplications cause symptoms and are, therefore, discovered in infancy or childhood, and that the condition is seldom found in adults (Nolan and Lee, 1953; Slomovitz, Cash, and Enzer, 1948). Ten of our 24 cases presented during the first 4 weeks of life; our oldest patient was 13 years of age. Abdominal duplications, unlike intrathoracic lesions, show a definite predilection for the male sex—70 per cent of our cases were boys. It has already been stated that abdominal duplications are not associated with abnormalities of the vertebræ.

Stomach.—Gastric duplications are rare. Keisewetter (1957) was able to collect 27 cases of gastric duplications from the literature. The single instance of gastric duplication in the series presented with symptoms and signs which suggested infantile pyloric stenosis—projectile vomiting, visible gastric peristalsis, and a large tumour palpable in the region of the pylorus.

Duodenum.—One of our 3 cases presented with signs and symptoms of high intestinal obstruction with bile-stained vomitus indicating obstruction below the ampulla of Vater. Another case complained of pain in the right hypochondrium and was jaundiced on admission. This child turned out to have a perforation of the cyst into the common bile-duct (*Case* 26).

Jejunum, Ileum, Colon.—The presenting signs and symptoms are usually those of intestinal obstruction—abdominal pain, vomiting, abdominal distension, failure to pass meconium or fæces. The high incidence of intestinal obstruction is noted by Fecher, Day, and King (1952).

An abdominal mass as the presenting symptom is supposed to be more rare. Dohn and Polvsen (1951) could find only 7 instances in a review of 124 intra-abdominal duplications. Intestinal obstruction can be caused by compression of the main channel due to fæcal impaction in a segment of duplicated gut, or by the duplication initiating a volvulus and by a cyst stretching the parent tube.

Two children in this series presented with intussusception; in one case the duplication was situated in the ileum and in the other in the cæcum. Four cases were admitted with peritonitis from perforation of a peptic ulcer in a duplication lined by gastric mucosa. Two of these children died. In 2 cases the duplication caused a volvulus, strangulation, and massive gangrene of the small intestine. Both were young infants and both died following massive resections.

Intestinal bleeding is often stated to be a common presenting symptom of tubular duplications, but in our series bleeding was uncommon. Only 3 cases presented in this way. *Case* 19 is particularly interesting:—

> This baby had recurrent bleeding from the rectum resulting in severe anæmia. At 9 weeks of age a laparotomy was carried out in another hospital, a cystic ileal duplication was resected with the adjacent segment of ileum. Twelve weeks later severe bleeding recurred, and she then came under our care.
>
> At a second operation a long tubular ileal duplication was found and resected. This case illustrates a point which is stressed by Gross and others (1952), that a second intestinal duplication can easily be overlooked.

Rectum.—Rectal duplications are rare and in those described in the literature signs of obstruction are the rule. A mass can usually be palpated on rectal examination. In 1 of our cases in this series rectal prolapse was the presenting symptom. The other case was most interesting; it presented as a tumour of the vulva simulating a teratoma. A segment of bowel, one end of which ended blindly in a nipple-like structure, the other extremity being open to the exterior, was adherent to the right labium majus. At operation the segment of bowel was adherent both to the rectum and vagina, but communicated with neither (*Case* 8, *Figs.* 474, 475).

FIG. 476.—*Case* 15. Cæcal duplication cyst.

TREATMENT

1. Mediastinal Duplications.—Formerly these cysts were often treated by marsupialization, which frequently resulted in secondary infection, fibrosis, scoliosis, and even death. Resection through a transpleural approach is now the method of choice.

2. Gastric Duplications.—As duplications are usually discovered in early childhood, partial gastrectomy is contra-indicated, but in adults this procedure is the method of choice. In childhood local resection is quite satisfactory unless the duplication involves the greater part of the stomach, when a partial gastrectomy might be obligatory.

3. Duodenum.—In our 3 cases it was possible to resect the duodenal duplication and to perform a duodenoduodenostomy. Resection and posterior gastrojejunostomy is not advisable (Gross and others, 1952). Gardner and Hart (1953) resected the duodenal wall between the duodenal lumen and the duplication cyst. This produces a large communal cavity and allows the cyst to drain freely into the lumen of the gut.

4. Jejunum and Ileum.—Resection of the duplication together with the adjacent bowel is usually performed. In very long tubular duplications resection may not be safe. Enucleation of a cyst, without tearing the normal bowel and impairing its blood-supply, is rarely feasible, and in fact Drennen (1931) stated that this is never possible.

In 1 of our cases (*Case* 21) we were, however, able to enucleate the cyst without injuring the bowel. Duplications of the ileocæcal angle will usually require resection of the terminal ileum, cæcum, and part of the ascending colon, with ileocolostomy.

FIG. 477.—*Case* 24. Tubular duplication of ileum.

5. Cæcum and Appendix.—Ravitch (1953) quotes many authors who had cases of double cæcum and appendix associated with a double colon. We have not had such a case.

6. Colon.—There was only 1 case with colonic duplication in this series (*Case* 1); in this the whole colon was duplicated. There were numerous severe associated abnormalities, as in cases reported by Ravitch (1953). Kratzer, Dixon, and Bargen (1951) reported a very interesting case of duplication of the colon and terminal part of the ileum associated with a parasitic twin. The double organs of the parasite were within a cyst situated in the epigastrium of the host.

Cases with double-barrelled colon are rare (Ravitch, 1953). Gross and others (1952) had 4 cases in their series of 68 duplications. They did not think that excision of the whole double-lumen colon was necessary. Only the lower segment should be tackled and a common lumen should be produced by resecting the intervening segment. In our case the whole colon was excised and the ileum pulled through an opening in the perineum. This child had a grossly shortened small intestine and finally died because of malnutrition.

7. Rectum.—Incision behind the rectum and dissection in the plane anterior to the sacrum is usually successful. Care has to be taken not to perforate the rectum. In cases with a double rectum and anus, one anus should be closed after the septum between the two lumina has been resected. The technique employed with the rare case of duplication presenting at the vulva consisted of its careful dissection off the vagina and rectum.

RESULTS

In this series 7 children died (25 per cent). The fatal cases were either very bad risks admitted with an advanced degree of peritonitis (*Cases* 7, 22, and 24), or were due to massive gangrene of the intestine (*Cases* 2 and 12), or multiple severe associated malformations (*Cases* 1 and 3). During the last five years with better pre-operative and post-operative management we have not lost a case.

SUMMARY

A series of 28 cases with duplications of the alimentary tract, seen during the last eight years, has been described. In this pædiatric surgical department, which serves a large population, this condition is therefore not too uncommon. No cases accidentally discovered at post-mortem examination have been discussed.

The embryology of the condition has been discussed and the main clinical features described.

Complete excision of the lesion is the method of choice.

There were 7 deaths due to peritonitis secondary to perforation, massive intestinal gangrene, or associated malformations.

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DIVERTICULAR DISEASE OF THE COLON

A REVIEW OF 258 CASES

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Diverticulosis coli is a condition that may remain silent or set in train a wide variety of unpleasant sequelæ which are all too familiar. The advent of antibiotics has placed a useful means of therapy in the hands of those surgeons who have long felt that a bolder approach is called for once the stage of diverticulitis is reached, and before the less manageable complications ensue. Unfortunately, the condition is often associated with advancing years and the general state of the patient may be a deterrent to radical surgery. However, this is not always so and there is a growing body of opinion in favour of elective surgery.

Surgery may also be demanded because of the added difficulty in managing those cases in which there is uncertainty as to the exact nature of the lesion, and the condition uppermost in our minds is carcinoma, which diverticulitis can so closely mimic.

This review is based on 258 cases admitted to the Western Infirmary, Glasgow, during the period 1945–56. We encountered 6 additional cases associated with carcinoma in the affected segment of bowel: these have been omitted for the sake of clarity but will be referred to later when we come to discuss the differentiation between diverticulitis and carcinoma of the colon.

AETIOLOGY

Opinion is divided as to the causation of colonic diverticula, and various factors may play a part in their formation. This review does not shed any further light upon the matter.

Congenital factors such as weakness in the musculature of the bowel wall may play a part in the aetiology, but it is probable that the condition is for the most part acquired. Upset of the normal sympathetic-parasympathetic balance of the nerve-supply to the gut may lead to muscular incoordination. The blood-supply to the bowel may be interfered with as a result of the degenerative processes of advancing years. Fatty infiltration of the bowel musculature in obese patients may also be a contributory factor (Illingworth and Dick, 1956).

Most colonic diverticula are of pulsion type and the possibility of alteration in pressure within the lumen of the bowel must enter into consideration of their formation. Thus, dietetic habits, flatulence, chronic constipation, and the abuse of purgatives may all lead to increased intralumenal pressure. These points are discussed by Sabeti (1948), who also includes focal infection as a possible cause.

The process commonly begins as small pouches where the terminal branches of the arterial arcades penetrate the circular muscle coat, and thus they are found mainly near the mesenteric border of the longitudinal muscle bands (Aird, 1957; Boyd, 1955; Edwards, 1954 a). As they develop and enlarge, the diverticula may become closely related to the appendices epiploicæ, and indeed it may be difficult to distinguish them at operation or autopsy because their presence is masked by pericolic fat and this difficulty increases in the presence of pericolic inflammation.

Table I.—Location of Disease in 258 Cases of Colonic Diverticula

Disease	Cæcum	Ascending Colon	Transverse Colon	Descending Colon	Pelvic Colon	Multiple Sites	Undetermined
Diverticulosis	0	0	2	4	30	16	6
Diverticulitis	0	0	1	7	136	42	14

SITE OF DISEASE

In the colon diverticula most commonly occur distal to the splenic flexure and the pelvic colon is by far the most common site. It must not be forgotten that several areas of the large bowel may be affected simultaneously and that sometimes these may be some distance apart. The sites of disease in the cases reviewed are shown in *Table I*. The pelvic colon was most commonly affected (64 per cent). The cæcum and ascending colon were conspicuously free of involvement in our series. Most authorities, among them Boyden (1950), quote comparable figures, but there are a few, such as Rankin and Brown (1930), who found the pelvic colon affected only in 29 per cent of their cases.

PATHOLOGY

As the wall of an established diverticulum is deficient or devoid of muscle-fibres its capacity for emptying itself must be poor, and in the distal colon, where the fæcal stream is more solid and more heavily infected, further complications may ensue. Episodic acute inflammatory changes may take place in the length of colon affected by diverticula and such inflammation may subside or:—

1. The inflamed pocket may rupture, producing peritonitis, local or general. Recently Ryan (1958) has drawn attention to the fact that perforation may occur even although there is free communication between the diverticulum and the bowel.

2. The inflammatory process may spread along the adjacent bowel wall leading to peridiverticulitis, with or without the formation of a frank pericolic abscess.

In the acute phase of inflammation there will be associated spasm in the affected length of bowel and this, added to any mechanical hold-up already present, will lead to a degree of intestinal obstruction, and in this series no fewer than 68 cases showed such signs. If the peridiverticulitis does resolve, fibrosis and scarring will take place in the affected bowel wall and these changes will tend to perpetuate the disease.

Acute pericolic inflammation may lead to the formation of adhesions, and fistulæ may form between the affected colon, small bowel, or bladder. Edwards (1954 b) considers the bladder as the organ most commonly involved in the spread of the disease and fistula formation. In the female a pericolic abscess may also discharge into the vagina, with the probable formation of a colovaginal fistula. Less often a pericolic abscess may track through parietal peritoneum to point in the anterior abdominal wall or the groin, and an external fistula may thus ensue. Still less commonly such a process may develop in the retroperitoneal tissues and point posteriorly.

Table III.—Ratio of Diverticulosis to Diverticulitis

Total Cases	Diverticulosis	Diverticulitis
258 (110 M.) (148 F.)	58 (23 M.) (35 F.)	200 (87 M.) (113 F.)

INCIDENCE

The incidence of diverticular disease appears to increase with the process of ageing, and it has been estimated that 5 per cent of all people over the age of 40 years are so affected, although asymptomatically (Wakeley, 1936).

In this series, *Table II*, the youngest patient was a male aged 24 years and the oldest a female aged 86 years. The average age of all patients reviewed was 64 years. The female sex showed a decidedly higher incidence, both of diverticulosis and diverticulitis, 148 out of a total of 258 cases (*Table III*). This is in contrast to the findings of Boyd (1955) and Illingworth and Dick (1956), who state that diverticula are twice as common in the male sex. However, Edwards (1954 a) finds that diverticulosis occurs with approximately equal frequency in the sexes but that diverticulitis is more than twice as common in the male.

Table II.—Age and Sex Distribution of Diverticular Disease

Sex	Number	Age-groups						
		20–29	30–39	40–49	50–59	60–69	70–79	80–89
M.	110	1	1	14	21	43	27	3
F.	148	0	2	5	33	49	51	8
Total	258	1	3	19	54	92	78	11

CLINICAL FEATURES

Pain.—In all but 33 of the 258 cases reviewed pain was a feature. In the majority the pain was of a dull, aching nature, often localized. However, 92 patients complained of colic spread over the lower abdomen, and in 72 cases the painful area was in the left iliac fossa and the findings were in keeping with the aphorism of 'left-sided appendicitis'. As many as 102 patients had pain in more than one site (*Table IV*).

Vomiting.—It is difficult to appraise the significance of vomiting in diverticulitis. This symptom was present in less than half the cases (*Table IV*), and, as expected, it occurred in those with acute intestinal obstruction. Much more often, however, vomiting occurred in the absence of other signs of obstruction and may have been reflex in origin.

Bowel Habit.—There is a wide variation in what patients regard as normal bowel habit, and an assessment is often a matter of conjecture. No distinctive feature appeared on analysis of the cases reviewed and there seemed little statistical difference in the number with regular bowel habits when compared with those with constipation or diarrhœa (*Table IV*). A small minority, 28 cases, gave a story of alternating constipation and diarrhœa, but this may have been due in part to the indiscriminate use of laxatives. The passage of mucus, with or without blood, was not a noteworthy feature in any patient.

Bleeding.—In 62 cases (24 per cent) a history of rectal bleeding was obtained (*Table IV*). This was associated with normal defæcation with the exception of 1 case in which a large spontaneous hæmorrhage occurred without other symptoms, as has been described by Fraenkel (1954). Hoar and Bernhard

Table IV.—Clinical Features in 258 Cases of Diverticular Disease

Pain			Site of Pain				Vomiting		Bowels				Bleeding		Weight Loss	
Absent	Colic	Dull ache	Hypo-gastrium	L.I.F.	R.I.F.	Other	+	−	Regular	Constipated	Diar-rhœa	Constipated and diarrhœa	+	−	+	−
33	92	133	76	72	25	102	107	151	73	94	63	28	62	196	41	217
	225															

(1954) have described colonic bleeding in its relation to diverticulitis and referred to 4 cases in which it was massive in quantity, with or without cramping pain: these authors emphasize that carcinoma of the colon is the more common cause of bleeding, either overt or occult, but not often a cause of a severe and alarming hæmorrhage. Noer (1955) recorded the incidence of rectal bleeding as being 10–30 per cent in his review of diverticulitis. As mentioned above, bleeding was the sole symptom in only 1 patient in our series, and we have, therefore, come to regard it as an uncommon manifestation of diverticular disease of the colon, a view which is shared by Gilchrist and Economou (1955).

Table V.—Recurrent Attacks of Symptoms

One Attack Only	Recurrent Attacks following Treatment		
	Conservative	Palliative	Radical
177	75	6	0

Loss of Weight.—Only 17 per cent of the cases investigated admitted loss of weight (*Table IV*). As anticipated, this was a feature of the more advanced stages of the disease, especially after complications, such as fistula formation, had developed.

Palpable Mass.—Accurate palpation of the abdomen was extremely difficult in many cases presenting with acute diverticulitis, especially if there was associated peritonitis. Tenderness and muscle guarding on occasion limited clinical examination, but nevertheless in no fewer than 48 patients a definite mass was palpable, most commonly in the suprapubic or umbilical area.

Fever.—A rise in temperature was found in approximately one-third of the cases in this series, and this was regarded as heralding one of the infective complications and indeed may be said to have coincided with the onset of symptoms.

Recurrent Attacks.—These occurred in 81 patients, 31 per cent (*Table V*), and extended over intervals ranging from one month to ten years. There was also a wide variation in the frequency of such episodes; for example, one patient had acute symptoms suggestive of diverticulitis on eight occasions within two years.

Obstruction.—The seriousness of this complication will be appreciated from our observation that 68 (26 per cent) of our series became obstructed to a greater or lesser degree (*Table VI*). This may be neurogenic or mechanical in origin, and the condition may present insidiously with minimal symptomatology, or be sudden in onset, with all the signs of complete hold-up. In the early stages of diverticulitis, the inflammatory changes may lead to disturbance of the normal nervous stimulation of the muscle-fibres of the bowel wall, resulting in spasm and upset of normal peristalsis in the inflamed segment of bowel. In the later stages of the disease, especially following repeated attacks, fibrotic changes set in, resulting in contracture of the gut and adhesion formation between the colon and loops of small intestine or neighbouring structures.

Peritonitis.—That this is an even greater danger is evidenced by our finding of a pericolic abscess in 51 patients out of a total of 258 cases reviewed, and a further 41 cases of perforation leading to diffuse peritonitis. A pericolic abscess is most often found in the left iliac fossa, as might be expected since the pelvic is usually the most affected segment. The diffuse or generalized form of peritonitis is often dramatic in onset, but occasionally the development of symptoms may be insidious. Rupture of an inflamed diverticulum may occur with the discharge of highly infected material into the general peritoneal cavity, and the development of paralytic ileus may be anticipated owing to the effect of toxic and inflammatory changes on the plexuses of Auerbach and Meissner. Hyperplastic and degenerative changes in Auerbach's plexus, following inflammation, were described by Celio (1952).

On occasion the clinical picture is confusing and the findings difficult to interpret, and laparotomy may be the only means of establishing a firm diagnosis, especially in the very ill case.

Fistula Formation.—This complication may result from spread of the initial disease or may follow operative intervention. As will be seen from *Table VI*, 10 cases developed a fistula, 7 of which came into the former category and 3 into the latter. The cases which developed fistula unrelated to operation comprised the following types: Enterocolic 2; Colovesical 3; Colovaginal 2. In the post-operative group of 3 patients the fistula was in each case external in type and had followed laparotomy and drainage. The 3 cases of colovesical fistula were females. This sex incidence is statistically insignificant, nor does it compare with the findings of Mayo and Blunt (1950 a), who noted a male preponderance of 5 to 1 in a series of 46 cases. They suggested that the uterus may act as a barrier to the formation of a colovesical fistula.

Table VI.—Complications of Diverticulitis (258 Cases)

Peritonitis and Acute Perforation	Pericolic Abscess	Obstruction	Fistulæ			
			Colovesical	Enterocolic	External	Colovaginal
41	51	68	3	2	3	2

MALIGNANCY

Carcinoma was suspected on clinical or radiological grounds, or even occasionally from the findings at laparotomy, in as many as 82 patients, but, as stated in our introduction, there were only 6 proven cases with an associated carcinoma and these have been omitted from the series. On the other hand, in another 3 patients, despite full investigation, a secure diagnosis of malignancy was not obtained.

What is the relationship between diverticulitis and malignancy? It may be that prolonged inflammation of the affected length of colon may lead to malignant changes, but proof of this is lacking and it is probable that the coexistence of the two lesions is incidental, and this was suggested by Devine and Devine (1948). We also note that Dukes (1958), in his paper on precancerous conditions of the large bowel, makes no reference to any relationship between diverticulitis and the subsequent development of carcinoma of the colon.

The clinician at times finds the differentiation of these two diseases baffling in the extreme since they can present so many features in common, and it is a problem which biopsy may fail to solve, because the specimen may be taken from an area not representing the true nature of the lesion and the presence of inflammation may make such examination difficult or even dangerous (Morton, 1952). We have no experience as yet of exfoliative cytology as a method of diagnosis, but are prepared to admit its possible advantages.

Even laparotomy, as most surgeons will agree, may leave the diagnosis in doubt. Most of these cases will require a colostomy and the true diagnosis reveals itself in the course of time.

SPECIAL INVESTIGATIONS

As has been inferred, the onset of diverticulosis or diverticulitis might be insidious and the symptom-complex obscure, but at this stage a *barium enema* often establishes the diagnosis. The cases that conform to the text-book description of 'left-sided appendicitis' are fairly easily diagnosed on clinical grounds alone. It is desirable, however, when the patient's condition permits, to carry out a confirmatory barium enema. This examination may show changes varying from spasticity, the presence of sacculations, to the 'saw-tooth' appearances of advanced diverticulitis. Occasionally a barium enema may not give the help anticipated owing to incomplete filling of the bowel. This situation is prone to arise where the clinical picture is confused by the suspected presence of carcinoma superimposed on diverticulitis. The administration of antispasmodic drugs may help in these circumstances, and Lloyd-Davies (1953) found that banthine bromide, 100 mg., given one hour before barium-enema examination aids relaxation of the gut and is thus of assistance in the difficult case. Barium-enema examination was undertaken on 212 patients in this series, and in all of these some abnormality was demonstrated in the colon.

Sigmoidoscopy appears to have a somewhat debatable value in the diagnosis of diverticulitis and its complications. However, it is felt that this examination should be undertaken especially when there is associated bleeding. Nevertheless in the acute stage of the disease sigmoidoscopy should not be lightly attempted because of the danger of trauma and possible rupture of the diseased area of the bowel, and such examination under an anæsthetic increases this risk. In these circumstances rigidity and narrowing of the gut due to either spasm or fibro-fatty change in its wall, contribute to make sigmoidoscopy a dangerous manœuvre, and diverticular openings are very rarely seen. These features have been discussed by Devine and Devine (1948). In this series sigmoidoscopy was performed on 63 cases, and of those only 7 showed an abnormality, and that was obstruction to the passage of the instrument.

Cystoscopy and Cystography would be expected to give additional information in those cases where there are urinary symptoms suggesting the involvement of the bladder by the colonic disease. Cystoscopy might reveal the presence of a colovesical fistula, but the orifice is often masked by the presence of infection and œdema. However, these findings would suggest such a complication and bacteriological examination of the urine would probably show the presence of organisms of bowel origin. The above findings might suggest that cystography would supply the necessary evidence, but failure to demonstrate a fistula on such radiological investigation would not preclude the presence of such a complication.

CONSERVATIVE TREATMENT

The great majority of cases suffering from diverticulosis or the milder forms of diverticulitis are relieved by simple medical measures, such as lubricating laxatives, or even bowel lavage and antispasmodic drugs may be worthy of trial. In this series a total of 194 cases were treated conservatively. As for the more specific forms of drug therapy, it will be appreciated that when the earlier cases in the series came under care only the sulphonamides were available. According to our records 111 patients received a chemotherapeutic agent or antibiotic either singly or in combination. In a number of these cases the drugs were used to minimize the bacterial flora of the gut prior to operation, but in many instances their administration was designed to reinforce other forms of conservative therapy. By and large, it is now our practice to use the broad-spectrum antibiotics, particularly if there is a threat of any complication. It is felt that the risk of avitaminosis and necrotizing enterocolitis following a therapeutic course of an antibiotic is minimal and may be disregarded provided the course is not too prolonged (Todd, 1955; Pettet, Baggenstoss, Dearing, and Judd, 1954; and Kay, Richards, and Watson, 1958). The results of medical treatment in our series will be considered later.

SURGICAL TREATMENT

The main indications are obstruction, peritonitis, and fistula formation. The suspected presence of malignancy demands surgical intervention. Repeated attacks of diverticulitis also provide a strong argument for extirpation of the disease before any of the above complications set in (Mayo and Blunt, 1950 b; and Sedgwick, 1951). Thus, the surgical care of this disease must be considered under two main headings: (1) Palliative; and (2) Radical Treatment.

1. Palliative Treatment.—In this series the palliative procedures employed were: (*a*) Laparotomy and peritoneal drainage; (*b*) Closure of a ruptured diverticulum, with or without drainage; (*c*) Cæcostomy; (*d*) Colostomy; (*e*) Enterostomy; (*f*) Entero-anastomosis; (*g*) Exteriorization. The cases in which a simple diagnostic laparotomy was

followed by closure of the wound are considered as having had conservative treatment.

A total of 53 patients was subjected to palliative surgery and in this group 23 (43 per cent) died. The data in *Table VII* give details as to the extent to which each of the above measures was used, and the associated mortality figures.

Table VII.—Cases treated by Palliative Surgery—Major Operation only Recorded

Sex	Laparotomy and Drainage	Deaths	Perforation closed ± Drainage	Deaths	Cæcostomy	Deaths	Colostomy	Deaths	Colostomy and Subsequent Closure	Deaths	Enterostomy or Anastomosis	Deaths	Exteriorization	Deaths
M.	10	4	2	1	0	0	5	3	3	0	1	1	1	1
F.	11	6	1	0	3	1	12	6	3	0	1	0	0	0
Total	21	10	3	1	3	1	17	9	6	0	2	1	1	1

Total No. of Cases—53 (M. 22; F. 31). Total Mortality—23 (M. 10; F. 13).

a. Laparotomy and Peritoneal Drainage.—This was the operation most commonly performed as a palliative measure and was undertaken on 21 patients (*Table VII*), but it is startling to find that this procedure was associated with a mortality rate of 50 per cent over the ten-year period of study. Nowadays, assuming a confident diagnosis of the true state of affairs can be made, a marked improvement in these results might be expected from the exhibition of antibiotics and possibly attention to the water–salt balance without having to resort to laparotomy and peritoneal drainage. Much of the current literature on diverticulitis suggests that there is no place for mere palliative surgery of this kind unless accompanied by some other measure, such as the formation of a colostomy. However, this view is not wholly shared by Cave and Alsop (1946) and Swinton (1951).

b. Closure of a Ruptured Diverticulum, With or Without Drainage.—Identification and closure of a ruptured diverticulum in the distal colon is usually impossible owing to the friable state of the tissues involved, but on occasion it has been achieved (Edwards, 1954 b). Ryan (1958) emphasizes that this method of treatment is unsound because invariably more than one diverticulum is inflamed,

Table VIII.—Cases treated by Multiple Palliative Operations

Case No.	Age and Sex	Colostomy	Colostomy plus Subsequent Closure	Cæcostomy	Laparotomy and Drainage	Result
S 4504/46	M. 37		(2) One mth. later after failure to resect mass (3) Colostomy closed 7 yr. later	(1) Later closed		Apparently well
S 2282/47	M. 58				(1) Laparotomy and drainage (2) Repeated 3 wk. later	Died
S 168/52	M. 68		(1) Transverse colostomy (2) Closed 7 mth. later		(1) Performed at time of transverse colostomy	Well 14 mth. later
51121	F. 56	(3) Transverse 1 mth. after second laparotomy and drainage			(1) Laparotomy and drainage (2) 6 mth. later	Well 9 mth. later
24287	F. 67	(2) Transverse colostomy 2 mth. later	(3) Colostomy closed 9 wk. later		(1) Developed external fæcal fistula 1 wk. later	Well 5 mth. later
7339	F. 55	(1) Six mth. after initial symptom			(2) Two mth. later	Died 4 mth. after second operation with external fæcal fistula
26588	F. 48				(1) Laparotomy (2) Laparotomy and drainage 8 yr. later	Well 2 mth. later
S 7/49	F. 56	(2) Permanent 2 wk. later		(1) Cæcostomy		At first thought to be inoperable carcinoma. Fairly well 5¼ yr. later

Total—8 cases (M. 3; F. 5). Deaths—2 (M. 1; F. 1).

and he advocates primary radical resection wherever possible, but diffuse peritonitis or acute obstruction are contra-indications to such a procedure.

c. Cæcostomy.—The indications for this procedure in the treatment of diverticulitis are ill defined. On 3 occasions cæcostomy was performed following

laparotomy and localization of the site of obstruction; 1 patient died. In a further 3 cases cæcostomy had been initially performed, but some 3–4 weeks later a colostomy was undertaken and therefore these 3 cases are included under this latter heading.

d. Colostomy.—This procedure was carried out in 17 cases as a palliative measure and 9 died (*Table VII*). It is most often required as an emergency measure in obstructive cases, but it may also be indicated where there is need to divert the fæcal stream from the inflamed area. Colostomy, moreover, might be regarded as a preliminary to resection of the diseased length of colon. A transverse colostomy was usually employed and this is the site advised by authorities such as Lloyd-Davies (1953), but occasionally it was possible to fashion a left iliac opening. This latter type of colostomy is limited in its suitability by the site of the disease or by fixation due to peridiverticulitis. On the infrequent occasion where there is perforation of a diverticulum in a localized area of inflammation it may be possible to utilize the perforated segment as a colostomy, but transverse colostomy promises the greater advantages both in theory and practice. Various modifications may be employed, but it is most desirable that the colostomy should be defunctioning in type. Also, if further surgery is anticipated, the opening should be made to the right of the middle colic vessels to avoid interference with the collateral circulation to the left colon.

We come now to the important question as to what should be done with the colostomy once the pressing need for it is long past. We believe that there is not a stock answer to the question.

On 6 occasions colostomy with subsequent closure without resection of the diseased bowel was performed. In 3 of these the follow-up notes extend over a few weeks only and no information is available regarding long-term results in these patients. In the remaining 3 cases which were followed up further attacks of diverticulitis occurred. Smithwick (1942) found that closure of an established colostomy, without resection of the affected segment, gave unsatisfactory results in 45 per cent of 36 cases. These findings are endorsed by the work of Todd (1955). Thus there is weighty evidence in support of resection of the diseased length of colon, a matter which is considered later.

e–g. Enterostomy, Entero-anastomosis, and Exteriorization.—These procedures may be described as 'last ditch' measures. *Enterostomy* was found to have been performed on 1 occasion and the patient died two days later. *Entero-anastomosis* was undertaken on 1 patient in whom the terminal ileum was anastomosed to the transverse colon because of several large diverticula involving the last few inches of the terminal ileum, cæcum, and ascending colon and causing apparent obstruction. This patient survived. *Exteriorization* of the affected segment of the large bowel was performed on 1 patient and death occurred within hours.

Table IX.—Cases with Multiple Operations—Palliative and Radical Combined

Case No.	Age and Sex	Colostomy	Colostomy with Closure	Laparotomy and Drainage	Exteriorization	Resection and Anastomosis	Result
42546	F. 66	(1) Colostomy	(2) Colostomy closed 10 yr. later			(3) Sigmoid colectomy 3½ yr. after closure of colostomy	Well
S 5797/52	F. 68		(2) Left iliac 5 mth. later (3) Closed 12 wk. later	(1) Laparotomy and drainage	(2) Exteriorization and resection of pelvic colon at same time as colostomy formed		Well 6 wk. later
71313	F. 67			(1) Followed by external fæcal fistula		(2) Resection of pelvic colon + fistula and end-to-end anastomosis 6 yr. later	Died 16 days post op.
S 2491/50	M. 59	(1) Left iliac colostomy				(2) Resection of sigmoid loop + colostomy 12 mth. later	Apparently well
15745	F. 62		(2) Transverse colon (4) Closed after resection		(1) Exteriorization of sigmoid	(3) Sigmoid colon resected	Well to date

Total—5 cases (M. 1; F. 4). Deaths—1 (F.).

Thus, it is obvious that the above procedures are unorthodox and apt to end disastrously, and are only to be considered as a desperate measure in a gravely ill patient. However, exteriorization of the segment of colon bearing the perforation, usually the sigmoid loop, is advocated as the procedure of choice by Aylett (1954) and Gilchrist and Economou (1955). It will be seen from *Table IX* that in 1 case exteriorization was performed as the first step of a planned resection.

Multiple Palliative Procedures.—*Table VIII* shows that 8 patients were treated by multiple palliative operations and of these 2 died, and it will be seen that some patients had three operations. In the majority of cases the initial procedure was laparotomy and peritoneal drainage, and in some it was necessary to take further steps to promote free drainage.

It may be concluded therefore that palliative measures are somewhat uncertain of success in the treatment of diverticulitis and its complications, and this has been the experience of Welch, Allen, and Donaldson (1953). Undoubtedly many such patients

are very ill and quite unfit for radical surgery at the time of the first palliative operation.

2. Radical Treatment.—Many now favour a more radical approach to the problem of diverticulitis. It has already been shown that if the condition was of such severity as to require the formation of a colostomy, further trouble may be anticipated if it is closed without resection of the affected bowel. At operation on cases who had a defunctioning colostomy performed several months previously for acute diverticulitis, the writers have been impressed by the fact that, although all active inflammation had settled, obvious diverticula were still evident and that they were full of fæcal matter. This impression was confirmed by examination of the resected specimens. Perhaps more impressive was the finding that invariably the lumen of the bowel distal to the colostomy was considerably diminished in calibre. Thus, restoration of the fæcal stream through such a length of colon would inevitably result in a flare-up of the acute inflammatory process.

The adoption of an earlier and more radical approach to the problem would undoubtedly avoid multiple operations and promise a greater measure of success. As early as 1950 Colcock advocated a staged radical approach—namely, colostomy followed by resection—and in 1953 Bartlett and McDermott appealed for radical surgery to be undertaken before the development of complications. Again, in 1956 Colcock advised radical surgical measures "in any patient with diverticulitis who has recurrent or persistent symptoms". Todd (1955) expressed a similar opinion. Judd and Mears (1955) advocated one-stage resection during a quiescent phase but advised multistage operations for the complicated case.

We need scarcely add that this is not the type of colonic surgery for the neophyte, and authoritative opinion stresses that the most suitable cases are those in which the disease is quiescent and localized to the pelvic and distal part of the descending colon. This should ensure a safe end-to-end anastomosis with the upper rectum or distal end of pelvic colon.

Whether or not resection and anastomosis is achieved at one operation or by staged procedures will depend upon the condition of the patient. In this series 11 patients were submitted to elective radical surgery and as far as is known no further symptoms arose which could be attributed to diverticulitis, but the time of follow-up is too short to allow of adequate assessment.

It goes without saying that the cases under review were prepared for operation with, at first, topical sulphonamides but now with broad-spectrum antibiotics. In practice a resection of colon for diverticulitis during a quiescent phase is often an easier technical procedure than a comparable operation for neoplasm. The sigmoid loop alone is affected in 85 per cent of cases which become complicated (Guy and Werelius, 1952), and resection of this length of bowel usually suffices. However, Ryan (1958) suggests that "diverticulotic" as opposed to "diverticulitic" bowel should be removed, and Gilchrist and Economou (1955) advised that the resection should include the descending colon if diverticula are present therein. The same care need not be taken to remove the relevant lymphatic glands and channels as when dealing with carcinoma, but on the other hand adhesions (perisigmoiditis) may make the operation difficult.

Table X shows that 11 cases were submitted to elective radical resection and a total of 8 patients had this major procedure undertaken without prior colostomy. For the purpose of this paper these cases have been classified under the heading of 'Immediate Resection'.

Table X.—Cases treated by Radical Surgery

Case No.	Age and Sex	Immediate Resection	Resection after Colostomy	Type of Resection
24521	F. 62	+		Resection of sigmoid and end-to-end anastomosis
42546	F. 66		+	Colostomy and 10 yr. later closure of colostomy. Three and a half years later resection of sigmoid with end-to-end anastomosis
7376	F. 73	+		Resection of sigmoid and end-to-end anastomosis
S 5797/52	F. 68	+		Resection of loop of sigmoid colon after exteriorization with anastomosis 12 wk. later
S 2749/49	F. 68	+		Abdominoperineal resection
74629	M. 57	+		Resection of rectosigmoid with end-to-end anastomosis
15745	F. 62		+	Laparotomy and exteriorization of sigmoid loop—transverse colostomy followed by resection
S 229/52	M. 80	+		Exteriorization of sigmoid and resection leaving ends as colostomy; anastomosis 3 mth. later
71313	F. 67	+		Laparotomy and drainage followed by external fistula at intervals for 3 yr. Six yr. after initial operation resection of descending colon and sigmoid with fistula included. Died 16 d. later
S 2491/50	M. 59		+	Left iliac colostomy followed by resection of colostomy and sigmoid colon. End-to-end anastomosis
S 3068/48	M. 72	+		Exteriorization of sigmoid loop as first stage of Paul-Mikulicz operation. Died within hours of operation
Total	11 Cases	8	3	2 Deaths

One of these 8 cases is of interest in that a laparotomy with drainage was done six years previously for diverticulitis and a fæcal fistula developed four weeks later. This healed spontaneously after some 12 months but broke down again and continued to discharge at intervals for three years. Resection of the descending and sigmoid colon, including the fistula, was performed, but unfortunately the patient died 16 days later.

The remaining 7 cases in this group were treated as under:—

Three cases: Resection of sigmoid colon with end-to-end anastomosis as a primary procedure.

Two cases: Resection of the affected loop of sigmoid performed immediately following exteriorization and the ends left as a colostomy. Formal closure of the colostomy, as distinct from a Paul-Mikulicz procedure, was undertaken three months later.

One case: Exteriorization and first stage of a Paul-Mikulicz resection of sigmoid colon performed. The patient's condition was so poor that no further surgery was possible and death occurred a few hours later.

One case: Abdominoperineal rectosigmoidectomy was performed on a female patient of 68 years in the mistaken belief that the lesion was malignant, and the true pathology became known only after operation.

Thus, it would appear that during the 10-year period under review there has been no standardized radical operation in the treatment of diverticulitis and the procedures undertaken depended upon varying factors. It will be seen from *Tables IX* and *X* that in only 3 cases was radical resection undertaken following a colostomy. Of these, 1 case is particularly instructive in that the colostomy had been established one year after the onset of the symptoms of diverticulitis, maintained for ten years, and then closed. This st p had been taken because sigmoidoscopy showed a narrowed but otherwise apparently healthy bowel. However, in the three years following closure of the colostomy there were recurrent attacks of diverticulitis and a palpable mass developed in the left iliac fossa. A barium enema confirmed the clinical findings of diverticulitis and resection of the sigmoid was then undertaken.

In the other 2 cases in this group the colostomy was formally closed during or after resection of the affected segment of large bowel.

RESULTS OF TREATMENT

(Excluding those with Carcinoma)

Although the cases which we have categorized according to the form of treatment adopted are not strictly comparable, the success or inadequacy of a particular line of management may be measured in terms of (1) the later course of the condition, or (2) the mortality incidence.

Conservative Treatment.—As shown in *Table XI*, there were 194 patients so treated, and of these 75 (39 per cent) (*Table V*) had further symptoms referable to the colonic disease and 11 (6 per cent) died. Ten of these deaths occurred during the first attack of diverticulitis and the remaining 1 during a second episode.

The average age of the group was 73 years and in 6 patients there was concomitant cardiovascular disease which doubtless was the primary cause of death. Another 2 cases had coexisting neoplastic disease in sites other than bowel, i.e., pancreas and ovary.

The remaining 3 deaths in this group were due to peritonitis and obstruction, and illustrative of these was a woman aged 53 years who was admitted in a moribund condition and quite unfit for operation. It was found that the primary cause of her illness was rupture of sigmoid diverticula with resultant diffuse peritonitis.

Table XI.—Type of Treatment and Related Mortality in Diverticulosis and Diverticulitis

Type of Treatment	Male	Female	Total	Mortality		
				Male	Female	Total
Conservative	84	110	194	2	9	11
Palliative	22	31	53	10	13	23
Radical	4	7	11	1	1	2
Total	110	148	258	13	23	36

Palliative Treatment.—All the 53 patients subjected to palliative surgery had some complication but were free of carcinoma (*see Tables VII* and *XI*). As many as 23 (43 per cent) died. It has already been stated that in some cases palliative operations were employed as a desperate measure and most of the deaths in this group occurred within 48 hours of the operation. In 20 cases the primary cause of death was peritonitis or obstruction, usually in combination. In addition, 2 patients died of cardiac failure and another had a fatal pulmonary embolism two weeks after operation.

Radical Treatment was carried out on 11 patients and 2 (18 per cent) died. For details we refer the reader to *Tables X* and *XI*. One of the deaths was that of a male aged 72 years, who had the sigmoid colon exteriorized and the first stage of a Paul-Mikulicz resection performed. Death took place within a few hours thereafter. The other was that of a female aged 67 years who died sixteen days after resection of an external fistula involving descending and sigmoid colon.

To summarize, the following broad principles may be said to govern the treatment of diverticular disease of the colon and its complications:—

1. The treatment of diverticulosis is medical. Regulation of the bowel habit is usually sufficient to control the condition and to prevent the development of diverticulitis.

2. Diverticulitis, in the early stages, may respond satisfactorily to medical or conservative measures such as the exhibition of chemotherapy or the broad-spectrum antibiotics and attention to bowel habit.

3. Recurrent attacks of diverticulitis or the development of complications such as obstruction, peritonitis, or fistula call for more energetic measures and surgical intervention becomes essential. Resection of the diseased length of colon with end-to-end anastomosis gives the greatest promise of complete relief of symptoms, but in the presence of peritonitis or obstruction this operation will probably require to be undertaken as a staged procedure, the

first step being a defunctioning colostomy, preferably situated in the transverse colon.

In conclusion, the analysis of the over-all results of treatment in this series of 258 patients lends support to the rising tide of opinion in favour of a more positive approach to the problem of diverticulitis. The implementation of such a policy calls for careful timing and selection, and it is our submission that radical treatment should be instituted when the disease seems likely to recur and before prohibitive complications set in.

SUMMARY

The clinical features of diverticular disease of the colon and its complications are presented. The similarity between this condition and carcinoma coli is stressed. The various special investigative procedures are briefly discussed. The over-all mortality in the total series was 14 per cent. This comprised 11 deaths in 194 cases treated by conservative measures (6 per cent); 23 deaths in 53 cases treated by palliative surgery (44 per cent); 2 deaths in 11 cases treated by radical resection (18 per cent).

A plea is made for consideration of radical surgery to be undertaken in the early stages of diverticulitis before complications ensue.

Acknowledgements.—We wish to express our gratitude to all the physicians and surgeons of the Western Infirmary, Glasgow, who allowed us access to their case records.

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SOLITARY DIVERTICULUM OF THE CÆCUM AND ITS COMPLICATIONS

BY D. B. BROWN AND W. F. TOOMEY

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A SOLITARY diverticulum of the cæcum is a rare condition. According to available records, Anderson (1947), Reid (1951), Parker and Serjeant (1957), the total number of cases reported is 114. The condition becomes a clinical problem when complications develop, e.g., acute inflammation or perforation. Naturally most, if not all, of the cases are wrongly diagnosed as acute appendicitis. At operation further difficulty may be encountered in establishing the true identity of the condition and not a few simulate cæcal carcinoma.

In 1937 Wilkie drew attention to the close similarity which may exist between acute inflammation of a solitary diverticulum and a malignant lesion of the cæcum as seen at operation. This difficulty was further discussed by Rosser (1944) and Cromar (1946), and again in 1950 Kirkman underlined the problem by referring to the number of cases that had been treated by right hemicolectomy.

The following case is illustrative of the diagnostic difficulties that arise:—

Case No. 155108. J. M. G., male, aged 65 years. Admitted on Nov. 25, 1958 to the Western Infirmary, Glasgow, with abdominal pain of two days' duration. The pain was dull in character, gradually increasing in severity, began in the hypogastrium and later moved to the right iliac fossa. There had been no vomiting.

There was a history of a similar attack six weeks previously, lasting one day.

On clinical examination there was tenderness and guarding in the right iliac fossa. There was no palpable mass and the temperature was 100·8° F.

Although the clinical signs were somewhat atypical it was thought that the patient was suffering from acute appendicitis and the abdomen was opened through a

right paramedian incision. The appendix was normal but there was a mass in the anteromedial wall of the cæcum, situated immediately distal to the ileocæcal valve. At the time the probability that the lesion might be an inflamed solitary diverticulum was in fact considered, but the resemblance to carcinoma was so close that it was decided to carry out a right hemicolectomy. Convalescence was straightforward, and the following pathological report showed that we were dealing with an acutely inflamed cæcal diverticulum.

Pathological Report.—The specimen consists of 10 cm. of small bowel with the cæcum and appendix. There is a 2·5-cm. diameter diverticulum in the cæcum with a 1-cm. os. The diverticulum has a necrotic wall and is filled with fæces. Microscopy confirms the diagnosis of a *diverticulum of the cæcum* which shows acute inflammatory changes. The epithelial lining has been lost and there is a very marked infiltrate of polymorphs. The serosal surface shows some inflammation also. No evidence of malignancy or of actinomycosis is seen.

AETIOLOGY

The site which a diverticulum occupies on the cæcum appears to be variable, but most of the recorded cases occur on the anterolateral or posterior aspects of the cæcum (Reid, 1951). According to Parker and Serjeant (1957) most authors regard it as congenital in origin, and Evans (1929) contended that it is derived from irregularities in the process of vacuolation of the intestinal endoderm during development of the cæcum. Others are of the opinion that a traction type of acquired diverticulum may result from adhesions (Dorling, 1945; Grace, 1940). In 1943 Schnug differentiated between 'true' and 'false' diverticula; in the former type all the bowel coats are present in the wall of the diverticulum, whereas in the latter one or more coats are absent. He also noted that 'true' diverticula are usually single and are almost invariably found in the region of the cæcum.

PERSONAL OBSERVATIONS

An inquiry into the clinical records of the Western Infirmary, Glasgow, over the ten-year period 1945 to 1956 yielded only 5 other cases of this condition. Each presented as an inflammatory complication and one had actually perforated. In order to gain some idea of the incidence of the condition we compared this number with the 258 cases of diverticular disease in other parts of the colon which were admitted over the same period, and arrived at an estimate of 2 per cent for complicated cæcal diverticulum.

Table I.—Details of Cases of Solitary Cæcal Diverticulum

Case Sheet No., Sex, Age	Clinical History and Findings	Pre-operation Diagnosis	Laparotomy Finding	Operation Performed	Result and Pathological Findings
64114 M. 47	Symptoms of acute appendicitis	Acute appendicitis	Gridiron incision. Appendix normal. Small well-defined mass in lateral wall of cæcum. Thought to be a lipoma	Mass removed and gap in cæcal wall closed and oversewn	Uneventful convalescence. Histology showed acute inflammation of cæcal diverticulum
S3840/53 F. 33	Colicky peri-umbilical pain 3/365, later spreading to R.I.F. Nausea. Temperature 99·6°	Acute appendicitis	Right paramedian. Appendix normal. Omentum attached to lateral wall of cæcum, where there was a mass. Pus expressed from hole in mass. Nature of condition uncertain	Right hemicolectomy	Convalescence straightforward. Histology: Acute inflammation of solitary diverticulum
S5821/46 M. 41	Colicky central abdominal pain of 18 hr. duration moving to R.I.F. No vomiting. History of previous attack	Acute appendicitis	Gridiron incision. Inflammatory swelling from lateral wall of cæcum, unrelated to appendix. Thought to be inflammation of solitary diverticulum	Outer coat removed and inner coat invaginated and oversewn. Appendicectomy	Convalescence uneventful
S4145/45 M. 43	Colicky pain in R.I.F. of 48 hr. duration. No nausea or vomiting. No history of previous similar attacks	Acute appendicitis	Gridiron incision. Inflamed mass in caput cæci. Thought to be solitary ulcer	Local resection, including appendix	Convalescence uneventful. Histology showed large diverticulum of cæcum with necrosis in parts. Ba. enema later negative
S1443/46 M. 31	Lower abdominal pain—48 hr. duration. No vomiting. No previous attacks. Tenderness and small palpable fixed mass in R.I.F.	Acute appendicitis	Gridiron incision. Perforation at the base of diverticulum of cæcum	Perforation closed and oversewn	Convalescence uneventful. Later Ba. enema—no abnormality

The clinical findings, management, and end-results of the additional 5 cases are presented in *Table I.* Taking into account the case cited in the introduction, it will be noted that there were 5 males and 1 female. The age range was 31 to 65 years, but Schnug (1943) found that the condition occurred equally in the sexes and the average age in his series was 40 years. The data given in the table show the presumptive diagnosis in each case was acute appendicitis, and it is interesting to note that a mass was palpated, and a small one moreover, in only 1 case. The lateral wall of the cæcum was the most common site of an acutely inflamed solitary diverticulum. As seen at operation the appearances of the lesion were variable. One was thought to be a lipoma, whereas two others were recognized as complications of an inflammatory process in the cæcum. It will also be noted that in one other case there was perforation of a solitary diverticulum. In 2 instances the lesion was thought to be a carcinoma. The

operation carried out varied with the circumstances in that, for example, local excision was performed of what was thought to be a lipoma; in another 2 cases conservative resection was undertaken; and the perforation was recognized, closed, and oversewn. However, it will be noted that in 2 patients, including the case described in the introduction, right hemicolectomy was undertaken in the belief that the lesion was malignant.

In all instances the results were satisfactory and convalescence was straightforward.

It must be conceded that the experience thus gained is unlikely to improve our diagnostic accuracy when the choice lies between acute inflammation of solitary diverticulum and appendicitis, but we feel that there are certain distinguishing features to be considered when the surgeon is faced at operation with the probability that he is dealing with a malignant lesion. These are briefly: young age-group, short history, and the infrequency of a palpable mass. All favour diverticulitis.

In conclusion, the fact remains that the occasion will arise when despite full consideration of all aspects of the case doubt will still remain as to whether or not the lesion is a malignant one. In such a circumstance we feel that right hemicolectomy is a rational procedure.

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RUPTURE OF PANCREATIC CYSTS

REPORT OF A CASE AND REVIEW OF THE LITERATURE

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PANCREATIC cysts, including pseudocysts, are rare and their complications even rarer. Rupture or perforation of the cyst is the commonest of these. When this occurs into the alimentary tract, temporary or permanent cure may result. On the other hand, rupture into the peritoneal cavity is usually associated with a high mortality. Some form of external drainage has generally been undertaken in the treatment of those cases which have come to operation. More definitive surgery has been attempted, however, in a few isolated instances. The case of intraperitoneal rupture now reported is a further example of this; recovery followed extragastric cystogastrostomy, the tear itself being utilized in the anastomosis. As reports of this complication are so infrequent, its diagnosis, prognosis, and treatment can only be discussed adequately against the background of a fairly extensive review of the world literature.

CASE REPORT

K. S., an Indian male, aged 47 years, was admitted to hospital on Jan. 8, 1957. He complained of epigastric pain, distension after meals, and anorexia for three months. There was no history of injury or previous abdominal pain.

ON EXAMINATION.—On physical examination a smooth, rather indefinite epigastric mass was palpated. A provisional diagnosis of pancreatic cyst was made and the following investigations were undertaken to confirm this, and to help assess his general condition.

Barium meal: Lateral view showed the stomach as a narrow strip with a concave posterior surface due to pressure of a large mass from behind.

Radiography of chest: Bilateral tuberculous cavitation.

Sputum: T.B. present.

E.S.R.: 73 mm./1st hour.

Hæmoglobin: 48 per cent (Sahli).

Stool: Hookworm ova present.

Urine: No sugar or albumin present.

Operation was postponed on account of the anæmia and gross lung pathology. The hookworm infestation (probable cause of the anæmia) was treated, and courses of oral and intravenous iron, streptomycin, and isoniazide started.

RUPTURE.—Three weeks after admission the epigastric mass, which had remained fairly constant in size, was felt to be larger and more definite. Further increase in size was confirmed the next day. The following morning the patient complained of sudden acute abdominal pain, and on examination the mass had disappeared, the abdomen was rigid and flat, and there was generalized tenderness and guarding. The pulse-rate was 110/min. and the blood-pressure 100/70 mm. Hg. It was concluded that the cyst had ruptured, and resuscitation was started while preparation was made for emergency operation.

OPERATION.—At laparotomy under general anæsthesia over 1·5 litres of fresh hæmorrhagic fluid were collected from the peritoneal cavity. A linear tear was found in the gastrocolic ligament extending for 4 cm. from near the spleen to the stomach, and along the greater curvature for a further 4 cm. The opening communicated with the interior of a large pseudocyst, which was adherent to the posterior stomach wall and the omenta. No bleeding point could be detected in the lining of the cyst. The lateral half of the rent could not be brought up for marsupialization as it was deep to the costal margin. It was closed with two layers of sutures. Opposite the remaining half of the tear an incision 4 cm. long was made in the adjacent stomach wall near the greater curvature, and these two openings were sutured together with continuous chromic catgut, forming an anastomosis between cyst and stomach. A single layer of sutures sufficed posteriorly as both structures were densely adherent, but

the usual two layers were used anteriorly. The abdomen was closed with drainage.

POST-OPERATIVE PROGRESS AND FOLLOW-UP.—Gastric suction and intravenous fluids were continued for 4 days. The drain was removed after 48 hours. Slight discharge continued from the drain site for several days but ceased spontaneously. Six weeks after operation a barium meal showed "hitching up" of the greater curvature of the stomach at the site of anastomosis in the erect picture, but the stoma itself could not be visualized, nor could barium be made to enter the cyst. The marked anterior displacement observed before was no longer present. The patient refused to continue treatment for tuberculosis as an in-patient, and was discharged on April 21. He attended irregularly as an out-patient, and when last seen 8 months after operation had no abdominal complaints. There was no clinical or radiographic evidence of recurrence of the cyst.

DISCUSSION

Survey of the Literature.—Sixty cases described as possible examples of rupture of a pancreatic cyst have been traced in the literature. The diagnosis can only be regarded as presumptive in some of those cases which progressed to spontaneous cure, as the records lack operative or autopsy details. A 'retrospective diagnosis' has sometimes been made by authors reviewing older reports.[1, 2] In other cases information as to the type of rupture is lacking.[3, 4] All these are worth noting, however, for to exclude them might lead to misconceptions regarding the incidence and prognosis of this rare complication.

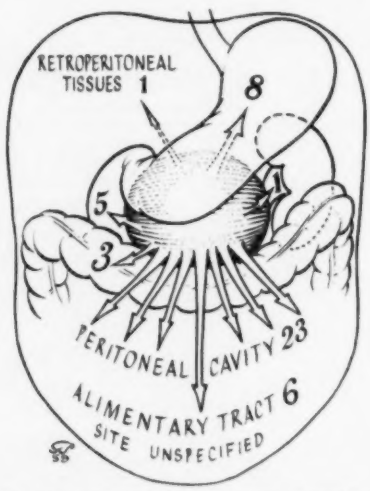

FIG. 478.—Rupture of pancreatic cysts. Probable site of rupture in 45 published cases comprising 47 perforations. Perforations which were the direct result of surgical interference have been excluded. In several cases a diagnosis of rupture into the alimentary tract was made, but there was no suggestion as to which organ had been involved.

Most authors have at least suggested the type of rupture, and from such reports it appears that perforation has occurred with equal frequency into a hollow viscus and into the peritoneal cavity. The site of rupture in 45 published cases (comprising 47 perforations) is shown diagrammatically in *Fig.* 478.

Hess and Rütte[5] estimated that about 5 per cent of pancreatic cysts ruptured. A personal estimate conforms very closely to this figure, but as reported cases alone were taken as an index of total incidence, the actual percentage is probably much lower, since it is reasonably certain that a higher proportion of ruptured than uncomplicated cysts have been recorded.

Rupture into a Hollow Viscus.—Rupture into the alimentary tract may lead to cure with disappearance of the cyst,[6-9] but sometimes this is only temporary or partial.[10, 11] Most of the unspecified cases may also be examples of such spontaneous drainage, but, even if they are included, about 50 per cent terminated fatally.[5, 12-14] Disappearance of the abdominal tumour has followed a bout of diarrhœa,[1, 2, 15, 16] and sometimes there have been several recurrences and remissions with severe diarrhœa preceding each remission.[17, 18] Hess and Rütte[5] and Opie[19] have suggested that discharge of contents may occur through pancreatic ducts communicating with the cyst. However, it has been demonstrated radiographically, at operation and autopsy that definite rupture has occurred into stomach, duodenum, and colon,[13, 20-24] and other probable cases have been reported.[14, 25-27] In two cases double rupture, involving more than one viscus, occurred.[5, 28] These, as well as several single rupture cases, presented with gross gastric or intestinal hæmorrhage. Some malignant cysts have eroded into the alimentary tract.[29, 30] Perforation of a cyst into the renal pelvis has been recorded, although the evidence for this was minimal.[31]

Intraperitoneal Rupture.—A pancreatic cyst may also rupture into the peritoneal cavity,[32-34] and this is usually associated with marked shock and a severe peritonitis.[29] Unlike rupture into the alimentary tract, it is doubtful whether any case of frank intraperitoneal rupture has survived without operation, although Hess and Rütte[5] and Körte[35] thought that this may have occurred. One or two of the cases recorded would perhaps be better described as delayed rupture of a lesser sac or pancreatic hæmatoma following traumatic laceration of the pancreas.[29, 36] Rupture is generally spontaneous, but trauma has been cited as an immediate cause in a few instances.[37] Palpation by the surgeon prior to laparotomy has ruptured several cysts.[38, 39] Pleural effusion in association with a pancreatic cyst has been attributed to rupture of the cyst with subsequent passage of the fluid from the peritoneal to the pleural cavity via the diaphragm hiatus![40] The dangers of aspiration of an uncomplicated cyst for diagnostic purposes or as a method of treatment have long been recognized,[41] and certain cases of delayed rupture following such a procedure have been cited sporadically in the older literature. Although some of these are rather doubtful,[42] others are well-authenticated examples of this complication.[43, 44] Cysts which recur following any form of operative treatment are also liable to rupture.[38, 45-47] Other rarer types of perforation, such as retroperitoneal rupture,[48] and rupture through the abdominal wall resulting in an external fistula,[16] have been recorded.

Diagnosis.—

Alimentary Tract.—Trias Pujol[7] describes the clinical features of rupture of a pancreatic cyst into the alimentary tract. These depend to some extent on the site of rupture. The full syndrome includes

pain, disappearance of the tumour, vomiting, hæmatemesis, diarrhœa, and melæna. There may be severe shock and the prognosis may be serious, but in the absence of gross hæm[illegible]age there may be no general upset. Barium studies may confirm the diagnosis in the less urgent cases.[21]

Peritoneal Cavity.—Koucky, Beck, and Todd[29] discuss the signs and symptoms of intraperitoneal rupture. They are generally those of an acute abdominal emergency in which a fluid-containing viscus has ruptured, and in the case reported closely simulated perforated peptic ulcer. Diagnosis was simplified by the fact that there had been opportunity to investigate the patient before any complication occurred. In cases where this has not been possible the history may be of assistance, and any reference to upper abdominal tumour or swelling should be noted. Absence of air under the diaphragm may help to exclude a perforation of the alimentary tract. Serum or urinary diastase levels may be raised in cysts of inflammatory or neoplastic origin.[49]

Treatment.—The case reported is a further illustration of the desirability of early elective operation once a diagnosis of pancreatic cyst has been made; rupture may occur if there is delay. The type of cyst may have some bearing on treatment, and Hess and Rütte[5] deal with the pathology of ruptured cysts. Although malignancy must be borne in mind, most ruptured cysts have been pseudocysts and the following remarks apply mainly to this type.

Laparotomy may rarely be indicated for gastrointestinal hæmorrhage following *rupture into a hollow viscus*, and the procedure to be adopted will depend on the findings. It may be possible to undersew a bleeding vessel at the margin of the perforation, but such has not been reported.

Operation is imperative for *intraperitoneal rupture* after adequate steps to counter shock have been instituted. Only about half of such cases have in fact come to surgery, but of these over 60 per cent survived the operation. This is a more optimistic estimate than that of Koucky and others[29] and Meyer, Sheridan, and Murphy,[50] who put the operative mortality at 66 per cent and 75 per cent respectively. Excluded from this analysis are 2 cases in which a localized abscess, rather than a general peritonitis, was related to a slowly leaking cyst; definitive treatment by cystogastrostomy or excision of the cyst was possible in both instances.[51, 52] Also excluded is a case where impending rupture with infarction of the cyst wall was averted by cystogastrostomy.[53]

External Drainage.—Marsupialization is probably the most suitable emergency operation for the majority of cases of intraperitoneal rupture. Koucky and others,[29] who with 6 cases on record have more experience of this complication than any other group of surgeons, have emphasized that when rupture occurs the simplest and most easily performed operation should be carried out. They are of the opinion that external drainage best fits these requirements. Some form of external drainage was used in 12 of the cases of intraperitoneal rupture under review.[29, 36, 54-58] Six patients recovered, but fæcal fistula complicated one of these. There have also been reports of gastric or duodenal fistulæ resulting from the pressure of tubes draining uncomplicated cysts.[59-61] Therefore, although simple drainage by suturing the rent around a tube may be the most rapid way of dealing with a ruptured cyst, it is perhaps inadvisable in cases where the tear is close to other organs. Formal marsupialization is preferable, but may occasionally be difficult for technical reasons such as are described in the personal case. Drainage following either of the procedures must usually be prolonged if recurrence of the cyst is to be avoided.[61]

Internal Drainage.—The question of more deliberate surgery should be considered in the light of the improved methods of resuscitation and the advances in anæsthesia which have made emergency surgery so much safer in recent years. Internal drainage is now regarded by many surgeons as the treatment of choice for uncomplicated pseudocysts. It has generally been avoided, however, as an emergency procedure for ruptured cysts. The indications for its use may now be broadened to include suitable cases of intraperitoneal rupture. The satisfactory outcome to the case reported, together with the evidence of 2 other reports in the recent literature,[28, 62] would support this suggestion. These 3 cases are possibly the only examples of ruptured cysts treated by internal drainage on record to date, and thus merit brief discussion. All presented different modifying features which favoured treatment by this method.

The personal case has already been described in detail. The fact that part of the rent was adjacent to the stomach facilitated extragastric cystogastrostomy. In one of the other cases[28] the operation was virtually elective. The patient was anæsthetized prior to laparotomy for a cyst which had recurred following previous cyst-to-end Roux-Y cystojejunostomy when the mass suddenly disappeared as the surgeon was palpating it through the relaxed abdominal wall. On entering the peritoneal cavity two perforations were found in a small friable area on the cyst wall. The previous anastomosis had closed and the defunctioned jejunal limb had separated from the cyst. This blind loop of the ready-made jejunal Y was anastomosed to the site of perforation, after trimming of the friable parts, forming a cyst-to-side Roux-Y cystojejunostomy. In both these cases the tear was dealt with and adequate drainage provided by the one procedure. The third case[62] differed in that no perforation could be found at laparotomy twenty hours after accidental rupture during liver biopsy, but the large pseudocyst had collapsed and the peritoneal cavity was full of cyst fluid. A small opening had presumably sealed off after the reduction in intracystal tension, and there was no need to deal with any open tear. Transgastric cystogastrostomy, by providing free drainage, proved adequate.

The relative merits of these various techniques of internal drainage are fully discussed in the literature.[63-65] As regards the method of drainage, however, it is pertinent to note that the most comprehensive analysis at present available[66] confirms the impression of many surgeons that for uncomplicated pseudocysts the morbidity is very much lower following internal drainage than it is following external drainage. The same can confidently be expected to apply to those ruptured cysts considered suitable to undergo the more elaborate operation.

Excision.—Total extirpation was successfully undertaken in one of the cases of frank rupture,[37] but was not considered in the case reported. It is an ideal, which even in uncomplicated cysts can only be attained at the cost of a high mortality, and is likely to aggravate shock and prolong the operation in an already acutely ill patient.

SUMMARY

A case of intraperitoneal rupture of a pancreatic cyst is reported, the literature reviewed, and treatment discussed.

Rupture is a rare complication of pancreatic cysts, and may occur equally often into a hollow viscus or into the peritoneal cavity.

Perforation into the alimentary tract may lead to cure of the cyst.

Frank intraperitoneal rupture is usually fatal unless operation is undertaken promptly.

External drainage has proved a life-saving operation in 50 per cent of cases in which it has been performed. It is the operation of choice in the seriously ill patient.

Only a few ruptured cysts have been treated by internal drainage, which was the method employed in the case reported. Although the results have been gratifying, such an operation should be reserved for favourable cases.

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A VESICAL CALCULUS CONTAINING METHYLENE BLUE

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THE following case is thought to be worthy of record on account of its unusual nature. Indeed, the writer has been unable to trace any previous report of methylene blue as a constituent of a urinary calculus.

CASE REPORT

H. H., a salesman, was 45 years of age at the time of his death. Until the age of 17 years he had led a normal life, and had been a successful athlete. At that time, however, he began to notice difficulty in gauging the fullness of his bladder and rectum, and in passing both urine and fæces. These symptoms developed progressively over the course of a year, at the end of which he had retention of urine with overflow incontinence, catheterization producing 2 pints (1·1 litres) of residual urine. He could only empty his bowels by means of self-administered enemata. Several doctors were consulted, but no firm diagnosis appears to have been reached.

When he was 21 years old a lumbar laminectomy and exploration was carried out, after which he was told that he had spina bifida, but no abnormality of the cord. Lumbar sympathectomy was also done at about this time, and was followed by an improvement in defæcation, though micturition remained unchanged. Suprapubic catheterization was tried, but proved unsatisfactory. It was therefore abandoned in favour of an indwelling Jaques urethral catheter, and this method the patient used for the remaining 24 years of his life. He himself passed, removed, and sterilized the catheters—which he changed every 2-3 weeks—and also gave himself bladder washouts containing hexamine and boric acid. To judge from his story, he contrived to avoid any overt urinary infection until a few months before his death. At 40 years of age tuberculosis of the right lung was found, and he was subsequently kept under observation at a Chest Clinic.

In late May or early June, 1956, he noticed pain in the right loin and the passage of dirty urine containing blood. Also he began to lose weight, and noticed a progressively enlarging mass over the left sternoclavicular joint, together with pain in the left lower back radiating down the lateral side of the left leg to the ankle. On June 8, 1956, he was admitted to St. Peter's Hospital, where he was found to have a large, radio-opaque vesical calculus (*Fig.* 479), a urinary infection due to *Bact. coli*, and a painful lump over the right sacro-iliac joint. He was transferred to Guy's Hospital (No. A 79199) on July 25.

ON EXAMINATION.—He appeared pale and ill, and two further hard subcutaneous lumps were found. These, like the two already mentioned, were thought to be metastatic tumours. There was flattening of the right upper chest, tenderness on suprapubic palpation, and the lower pole of the right kidney could be felt. On rectal examination the large vesical calculus was palpable above the prostate. There was flaccid weakness of the left quadriceps femoris, the knee-jerk on that side being diminished.

Chest radiography showed multiple rounded opacities in both lung fields, and there were also osteolytic changes in the left ilium suggestive of metastases. Biopsy of a subcutaneous nodule from the abdominal wall was reported as showing a metastatic deposit of poorly differentiated squamous-cell carcinoma (Dr. S. J. De Navasquez: SS 1888/56). Thereafter analgesic drugs were given until the patient's death on August 20.

NECROPSY FINDINGS (G.H.P.M. 256/56).—

Spina bifida occulta: sacral termination of spinal cord.
Vesical calculus, large, and squamous metaplasia.
Carcinoma of bladder: extensive metastases.
Acute cystitis: bilateral acute pyelonephritis.
Pulmonary tuberculosis (inactive): emphysema.

There was severe spina bifida occulta in the lower sacral region (*Fig.* 479). Instead of ending at its usual level, the spinal cord, which was at least 55 cm. long, entered the sacral canal to end in the region of the second sacral vertebra, where it was adherent. (A case almost identical in this respect has been described by Reimann and Anson, 1944.) The primary tumour, a fungating discoid mass, 55 × 45 mm., arose from the left side of the upper posterior wall of the bladder, which contained only the calculus and a little foul-smelling, blood-stained, purulent fluid. Metastases were present in lymph-nodes, lungs, liver, right adrenal, and many bones, causing pathological fractures of the sternum and of several ribs.

FIG. 479.—Radiograph of the pelvis and lower lumbar spine on July 27, 1956, showing the very large and densely radio-opaque vesical calculus, the sacral spinal bifida, the old lumbar laminectomy, and the tip of a catheter.

Histological Findings.—Microscopy of the tumour (7 blocks) showed it to be a carcinoma—both papillary and solid in structure—which had infiltrated the full thickness of the vesical wall. Its component cells varied in type from one part of the tumour to another. Some portions of the papillary growth consisted of transitional-cell carcinoma, but, for the most part, the cells here bore little resemblance to those from which they had taken origin. They were not, however, completely anaplastic, many showing clear evidence of squamous differentiation, both in their form, and by the formation of keratin within the cytoplasm of some individual cells. The solid and infiltrating portions of the tumour—though anaplastic in parts—showed for the most part a similar tendency to squamous differentiation, which was reproduced in all the metastases examined microscopically.

The surface of the tumour was partly necrotic. The debris enmeshed in its irregularities included many minute rounded nodules of calcific material, numerous bacteria—chiefly Gram-negative rods, and neutrophil polymorphs.

The Calculus.—This had the shape of a flattened ovoid; it measured 69 × 56 × 40 mm. and weighed 165 g. Outwardly it was principally white in colour, though underlying sandy-coloured material was visible in some

parts (*Fig.* 480). It was bisected at once in the post-mortem room with a steel hacksaw, and proved to cut fairly easily, its substance being moist throughout. After a little cutting the sawdust suddenly became a brilliant pale (Cambridge) blue in colour, but when bisection was complete no material of comparable colour was to be seen in the cut surfaces. Instead, a band of a dull greenish-bluish-grey was seen to encircle the centre of the stone, just beyond midway from the centre to the outer surface. During the next 10 minutes a dramatic change occurred

FIG. 480.—Colour photograph, showing both the cut and external surfaces of the calculus, taken after the change of colour —consequent upon oxidation of the methylene blue—was complete. The concentrically-laminated structure of the stone is well shown. (Some pale blue sawdust has become embedded in the outermost strata, and has also caused slight blue staining of the external surface over a small area.)

in both cut surfaces. The band just described rapidly changed in colour until it was a deep royal blue, as did the very centre of the calculus; while at the same time the part between took on a lighter blue. The portion peripheral to the deep blue band showed no change, remaining a dull greyish-white. The appearance of the cut surface after full development of the colours is shown in *Fig.* 480, which also demonstrates the concentrically laminated structure of the stone. Its centre contained no apparent foreign body or other definite nucleus, and was indeed more friable than the outer layers. Except for some slight lightening of both shades of blue as the stone became dry, the colours have not changed during the subsequent two and a half years.

The calculus therefore contained a substance which—colourless or all but so when the stone was first bisected —had been rapidly changed to a blue colour by some physical or chemical influence acting upon it during or immediately after the sawing. It was thought that the transformation was most probably due to the conversion by atmospheric oxygen of methylene blue from its reduced, colourless, or 'leuco-' form to its blue oxidized form.

Two samples of the stone were submitted to chemical analysis. The first, from the greyish-white outer strata, proved to be chiefly calcium phosphate, with some calcium oxalate and small amounts of magnesium ammonium phosphate and calcium carbonate. The second, derived from the outer and intermediate blue zones, also consisted chiefly of calcium phosphate with lesser amounts of magnesium ammonium phosphate and a trace of calcium oxalate. Its blue component was found to have the following properties: first, it was soluble in water, ethyl alcohol, and chloroform; secondly, it could be reversibly decolorized by suitable reducing conditions achieved by boiling a solution in the presence of glucose and sodium hydroxide—when this mixture was subsequently cooled and oxygenated by vigorous shaking the blue colour rapidly returned. Finally, an alcoholic solution was examined spectrophotometrically and was found to have a main absorption peak at a wave-length of 6660 A.U. and a much lesser peak between 6000 A.U. and 6200 A.U. A simultaneous comparison was made with alcoholic solutions of known methylene blue and of a pill of the variety thought to have been its source in the present case. In both instances the absorption curves obtained were closely similar to that of the blue substance from the calculus. In the light of all these findings it was concluded that this substance was methylene blue.

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SUMMARY

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2. The centre of the calculus contained much methylene blue, identified by its solubility, its reversible reducibility to a colourless compound, and its spectrophotometric absorption curve. This was principally in the reduced, colourless or 'leuco-' form when the stone was first removed from the heavily infected bladder and bisected. Following exposure to atmospheric oxygen it became converted to the blue oxidized form within a few minutes.

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parts (*Fig.* 480). It was bisected at once in the post-mortem room with a steel hacksaw, and proved to cut fairly easily, its substance being moist throughout. After a little cutting the sawdust suddenly became a brilliant pale (Cambridge) blue in colour, but when bisection was complete no material of comparable colour was to be seen in the cut surfaces. Instead, a band of a dull greenish-bluish-grey was seen to encircle the centre of the stone, just beyond midway from the centre to the outer surface. During the next 10 minutes a dramatic change occurred

FIG. 480.—Colour photograph, showing both the cut and external surfaces of the calculus, taken after the change of colour —consequent upon oxidation of the methylene blue—was complete. The concentrically-laminated structure of the stone is well shown. (Some pale blue sawdust has become embedded in the outermost strata, and has also caused slight blue staining of the external surface over a small area.)

in both cut surfaces. The band just described rapidly changed in colour until it was a deep royal blue, as did the very centre of the calculus; while at the same time the part between took on a lighter blue. The portion peripheral to the deep blue band showed no change, remaining a dull greyish-white. The appearance of the cut surface after full development of the colours is shown in *Fig.* 480, which also demonstrates the concentrically laminated structure of the stone. Its centre contained no apparent foreign body or other definite nucleus, and was indeed more friable than the outer layers. Except for some slight lightening of both shades of blue as the stone became dry, the colours have not changed during the subsequent two and a half years.

The calculus therefore contained a substance which—colourless or all but so when the stone was first bisected —had been rapidly changed to a blue colour by some physical or chemical influence acting upon it during or immediately after the sawing. It was thought that the transformation was most probably due to the conversion by atmospheric oxygen of methylene blue from its reduced, colourless, or 'leuco-' form to its blue oxidized form.

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NEW CONCEPTS ON AMŒBIC ABSCESS OF THE LIVER

By MILROY PAUL
PROFESSOR OF SURGERY, UNIVERSITY OF CEYLON

MATERIALS AND METHODS

THIS paper is based on experiences with the management of cases of amœbic hepatitis and amœbic abscess of the liver over a period of thirty years. A consecutive series of 196 cases of amœbic hepatitis and 203 cases of amœbic abscess of the liver under the writer's care at the General Hospital, Colombo (1936–59), is reviewed. The findings in those cases in this series in which the lesion was exposed at an operation or displayed at a post-mortem examination were recorded by the writer soon after each observation, and these findings are used to formulate a new concept of the lesions of amœbic hepatitis and of amœbic abscess of the liver.

The clinical features of these cases have been correlated with the finding of abscesses at different sites in the liver, and clinical syndromes characterizing the abscesses at different sites in the liver have been defined. Cases are recorded in which recognition of these clinical syndromes permitted accurate localization of abscesses in the liver.

PART I

Amœbic Hepatitis: Does it Exist?—The term 'amœbic hepatitis' is widely used and cases of amœbic infections of the liver are classified as being either amœbic hepatitis or amœbic abscess of the liver. What does the term 'amœbic hepatitis' mean? The term was introduced by Rogers (1922) to comprise those cases in which an infection of the liver by *Entamœbæ* had caused an inflammation which had not proceeded to the stage of suppuration. Rogers described the lesion as a diffuse inflammation of the whole liver caused by invasion of numbers of *Entamœbæ* which were filtered from the portal bloodstream in the smaller interlobular veins of the liver.

Although Rogers's work on amœbic lesions in the liver was based on observation of a vast amount of post-mortem material, there is no record by Rogers of the demonstration of a liver diffusely inflamed, with *Entamœbæ* enmeshed in the interlobular veins of the whole extent of the liver. Rogers's concept was based on the clinical findings in cases in which a painful and tender liver recovered completely on treatment with amœbicidal drugs. Is this evidence sufficient to establish the entity of the lesion postulated by Rogers? Although Rogers's concept has been widely accepted, the lesion described by him has never been demonstrated either at an operation or at a post-mortem examination. Does this not raise the question of whether the lesion described by Rogers really exists?

In order to decide this question, the clinical findings in the 196 cases of amœbic hepatitis in this series were examined. These cases had had the systemic disturbances of an inflammation and pain and tenderness of the liver. The pain and tenderness of the liver were, however, localized to a small area within the intercostal spaces overlying the right lobe of the liver. None of the cases had pain and tenderness of the whole liver. The evidence of these cases suggests that amœbic hepatitis is a localized lesion in the right lobe of the liver near its lateral surface. It is only lesions at this site that draw attention to themselves by pain and tenderness. Lesions at other sites in the liver do not cause pain and tenderness in the early pre-suppurative stages, and such lesions are latent and would not be recognizable as cases of amœbic hepatitis.

Although there is no evidence to warrant acceptance of Rogers's concept of a diffuse inflammation of the liver, the term 'amœbic hepatitis' is a useful one to classify these cases with pain and tenderness over the liver, which recover completely on treatment with amœbicidal drugs. The lesion could be at the pre-suppurative stage or it could have suppurated and then resolved completely on treatment with amœbicidal drugs. Acceptance of the concept of amœbic hepatitis as a localized lesion and not a generalized inflammation leads to the conclusion that amœbic lesions in the liver are from the onset isolated at one or at most a few foci only, that the lesions are the result of the lodgement of the *Entamœba* at these isolated sites and that the rest of the liver is unaffected. The lesion enlarges from its site of origin and involves more and more liver by direct extension.

Amœbic Abscess of the Liver.—The production of pus by amœbæ in a liver is a strange phenomenon. Pus is produced by the growth of micro-organisms in the tissues. In a liver abscess invasion by *Entamœba histolytica* results in the formation of pus. Is this a peculiar form of suppuration, or is it no different from the suppuration produced by micro-organisms?

The Significance of the Finding of Amœbæ in an Abscess of the Liver.—Amœbæ were found in the capillaries and tissues adjoining the wall of a liver abscess by Koch (1883) and in scrapings of the walls of liver abscesses by Kartulis (1887). The idea of suppuration being caused by amœbæ was so unusual that Kartulis believed that the suppuration was due to micro-organisms carried to the liver from the colon by the amœbæ. Micro-organisms are, however, absent in some cases of liver abscesses teeming with amœbæ, and in such cases it is evident that the amœbæ must be the agents of suppuration! Amœbæ in the walls and granulations of a liver abscess could not be harmless wanderers in the tissues of the body. They must be pathogenic (Councilman and Lafleur, 1891). The finding of amœbæ in an abscess of the liver gives proof that amœbæ are the cause of that abscess.

Would Amœbæ be found in every Amœbic Abscess of the Liver?—Amœbæ are not found in the pus or in scrapings from the walls of some abscesses of the liver. It is possible that though there were no amœbæ in the particular samples examined, there may have been amœbæ at other sites in the abscess. However, examinations undertaken by us of amœbic abscesses coming to post-mortem failed to locate amœbæ anywhere in the

liver of some of the cases. As there were also no micro-organisms in the pus of these cases, it was evident that amœbæ may die out after producing an abscess in the liver. There may therefore be no amœbæ anywhere in a liver which has been invaded by amœbæ at an earlier date.

The Significance of the Finding of Micro-organisms in the Pus of a Liver Abscess.—Micro-organisms are found in the pus of liver abscesses, and in such cases there may or may not be amœbæ in the abscesses. If amœbæ are present it is probable that the abscess was produced by the amœbæ, and that the micro-organisms secondarily invaded the abscess. It is unlikely that amœbæ would secondarily invade a liver abscess which had been produced by micro-organisms reaching the liver. Even in abscesses in which amœbæ are absent, the abscess could still have been one in which the amœbæ had died out after producing the abscess. The finding of micro-organisms in the pus of a liver abscess does not therefore establish that the abscess was produced by the micro-organisms.

Are all Liver Abscesses Amœbic Abscesses?—The large abscess of the liver is common only in countries where amœbic dysentery is rife. In other countries abscess of the liver is a rare condition, and rare though it be in these countries, the abscesses occur there only in persons who have previously been to countries where amœbic dysentery occurs. The only pyogenic lesions occurring in the liver in persons who have not been exposed to the hazard of contracting an amœbic infection of the colon are suppurative cholangitis and suppurative pylephlebitis. The suppuration in these lesions is confined to dilated biliary or venous channels. There is no appreciable extension of suppuration into the liver substance from the walls of these channels.

The review made of the geographical distribution of the cases of liver abscesses establishes that all cases of liver abscesses are amœbic, whether amœbæ are found in the abscess or not, and whether pyogenic micro-organisms are found in the abscess or not. The large abscesses of the liver described by Hippocrates must have been amœbic abscesses.

How do pyogenic micro-organisms get to a liver abscess? Pyogenic micro-organisms do not form abscesses in peoples who have never been exposed to the risk of sustaining an amœbic infection of their colons. It is therefore likely that the micro-organisms occasionally found in the pus of liver abscesses have been carried to the abscess from the colon by the amœbæ themselves. Micro-organisms must on occasion reach the liver by other routes, but the evidence of the geographical incidence of liver abscesses negatives the formation of large abscesses in the liver from such infections. Reports of the formation of such abscesses by pyogenic micro-organisms are invariably from countries where amœbic dysentery is rife (Rothenberg and Linder, 1934; Ochsner, de Bakey, and Murray, 1938; McFadzean, Chang, and Wong, 1953).

Is the Pus from an Amœbic Abscess of the Liver different from the Pus formed by the Pyogenic Micro-organisms?—As the *Entamœba histolytica* is the only organism apart from the pyogenic micro-organisms capable of forming pus in the tissues, it would be natural to expect that there might be some differences in the modes of suppuration, and that the pus in an amœbic abscess of the liver would be different from the pus formed by pyogenic micro-organisms. This expectation receives some measure of support from the practice in tropical countries of recognizing amœbic abscess pus by its characteristic colour and consistence. Amœbic abscess pus is either chocolate or cream coloured, with the thick viscosity of a heavy grade lubricating oil. Pus with these features is so very different from pus from other suppurative states in the abdominal or peritoneal cavities that surgeons can rely on recognition of amœbic abscess pus to indicate that the pus comes from the liver. Although such recognition is of value, the pus of an amœbic abscess is not different from the pus formed by pyogenic micro-organisms at other sites. Creamy pus with the consistence of a heavy grade lubricating oil is to be found in many abscesses of staphylococcal origin. A perinephric abscess has creamy pus very like the pus in a liver abscess. The chocolate colour of liver abscess pus is not due to autolysis of the brown liver. Liver tissue is really white, not brown. The brown comes from the blood within the liver. A liver drained of blood is white. That the chocolate colour of the pus is due to admixture with fresh blood is shown by examination of such pus under the microscope. Chocolate pus always contains red blood cells of normal size and contour, and these could only have come from a recent hæmorrhage. Liver abscess pus also gives evidence of being a mixture of blood and pus in the streaks of chocolate or raspberry jam in creamy pus. Chocolate pus is not distinctive of a liver abscess. Chocolate pus indistinguishable from liver abscess pus is found in hæmatoceles where blood has freely mixed with the pus.

Liver abscess pus is not always thick. If bile flows into the abscess cavity, the abscess contains thin bile fluid. Sometimes even when there is no bile, the pus is thin and watery with flakes of lymph in it. Liver abscess pus is therefore not different from the pus formed by micro-organisms. Nevertheless, the pus has features which are different from the pus found in many of the common intra-abdominal and pleural suppurations, and these features are of value in recognizing pus found in the abdominal or pleural cavities which come from a liver abscess.

The Cellular Content of Amœbic Abscess Pus.—Examination of unstained films of the pus under the microscope shows granular debris, red blood-corpuscles, and white cells. The red blood-corpuscles are usually of normal size and contour, but there may be a few crenated cells. The *Entamœba histolytica* is found in the pus in a minority of the cases. Stained films of the pus show that the white cells are so degenerate that they are only just identifiable as polymorphonuclear or mononuclear cells. The mononuclear cells are not recognizable as mononuclear leucocytes or tissue cells. There are sometimes clumps of cells. These cells must be from the liver, but they are so degenerate that they are not identifiable as liver cells. Stained films of the pus may show micro-organisms in some of the cases.

There is therefore nothing in the cellular content of amœbic abscess pus to distinguish the pus from

the pus produced by pyogenic organisms. Cheatle (1923) defined pus as the result of the interaction of an external excitant upon living tissues causing death of tissues and emigration of leucocytes and of serum. All these are found in an amœbic abscess.

The Morbid Anatomy of a Liver containing an Amœbic Abscess.—The amœbic abscess is usually a spherical lesion larger than an orange but with a wall only a few millimetres thick consisting of granulation tissue. There is little or no fibrosis. The content of the abscess is usually fluid pus, but on occasion it is filled with a semi-solid yellow slough.

FIG. 481.—Multiple abscesses of the liver. Histological sections of the liver showed no change in the liver beyond the walls of the abscesses. There were many abscesses (2–3 mm. in diameter) in this liver. Histological sections showed that the liver was normal beyond the walls of these abscesses. Sections from the walls of the large abscesses showed a few amœbæ.

A liver may contain one, two, or even three such large abscesses (*Fig.* 481). The liver beyond the walls of the abscesses is normal in appearance and histological examination shows no change in the liver tissue beyond the abscesses. Investigations carried out by us on livers coming to post-mortem showed that there were no amœbæ beyond the walls of the liver abscesses and the liver showed no cellular infiltrations beyond the walls of the abscess. In a few cases there were, in addition to one or more large abscesses, myriads of small abscesses each a few millimetres in diameter. Even in such cases, the liver beyond the miliary areas of suppuration was normal. This evidence suggests that each amœbic abscess comes from the lodgement, survival, and proliferation of a single amœba in the liver, and that the abscesses are all of the same duration. There is no evidence of invasion of the whole of a liver by showers of amœbæ causing diffuse inflammation of the liver, and then suppuration at certain foci in a diffusely inflamed liver.

The Healing of a Liver Abscess.—It is remarkable that routine post-mortems in tropical countries do not reveal old-standing chronic cavities in the liver from a burnt-out abscess of a past period. Some liver abscesses are so large that one would expect that healing would cause gross fibrosis and distortion of the liver. Such distortions are never seen. In one case examined post mortem there were a few superficial scars which could have been the site of old abscesses, but there was no distortion of the shape of the liver. The gross destruction of an appreciable volume of liver tissue by an amœbic abscess must therefore be repaired so completely that there is no trace of the cavity which had been occupied by the abscess.

PART II

How is a Liver Abscess detected?—A liver abscess becomes clinically evident when it causes general constitutional disturbances together with symptoms and signs indicative of a local inflammation in the liver. The general constitutional disturbances are the predominant features of the clinical picture and they determine the patient to seek advice. Although the fever with chills and rigors, the anorexia, and the profound emaciation cause a rapid deterioration in the condition of the patient, there is no hint in these disturbances that the causative lesion is in the liver, and a patient could die without the lesion in the liver being detected.

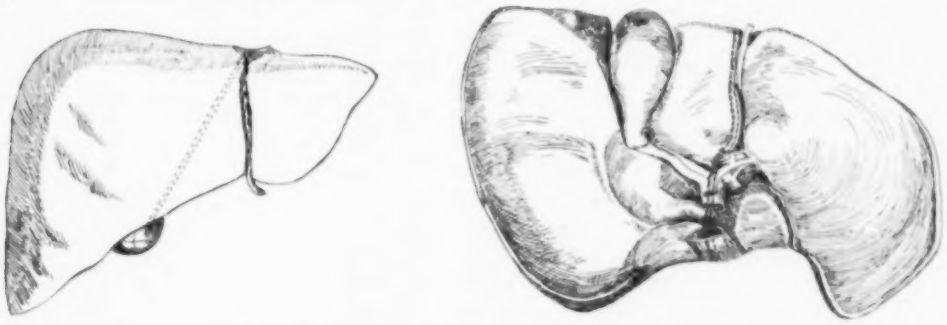
The diagnosis of a liver abscess is made from symptoms and signs indicative of a local lesion in the liver. These symptoms and signs are not obtrusive and a careful clinical investigation is needed for their elucidation. Little attention has been paid in the literature to this aspect of the problem. The descriptions given of the clinical findings in amœbic abscess of the liver are the symptoms and signs found in the common case where the abscess presents under the right chest wall. No note has been taken of the very obvious proposition that the symptoms and signs of a local lesion in the liver would be dependent on the position in the liver of the abscess. A review of the clinical findings in the series of cases reported on in this paper led to the recognition of clinical syndromes characterizing abscesses at different sites in the liver. The clinical syndromes permit precise recognition of the site of an abscess in the liver, and this permits a more accurate plan of treatment for the abscess. A survey of the clinical syndromes on which a diagnosis was based led to the realization that these different clinical syndromes are produced only when an abscess in the liver reaches to one of the surfaces of the liver. The abscess has also to be of appreciable size to affect a sufficient area of the surface of the liver to cause localizing symptoms and signs. It became evident that the abscess causes no local symptoms or signs till it reaches the surface of the liver, and a diagnosis of liver abscess is not possible till this happens. How long would this take? The

evidence of cases coming for treatment shows that patients have evidence of general constitutional disturbances for no longer than a few weeks before seeking advice, and when they do seek advice there are already localizing symptoms and signs of a lesion in the liver. The abscess of the liver must therefore have reached the surface of the liver within these few weeks of the onset of symptoms of ill health. The rapid deterioration in the general condition of patients within these few weeks gives evidence that abscesses of the liver are acute and that they increase rapidly in size. These deductions from the clinical evidence are supported by observations made of the morbid anatomy of these abscesses when they are exposed at operations or at post-mortems. The wall is made up of granulation tissue of a few millimetres' thickness, with very little evidence of fibrosis.

The line best demarcating the abscesses of the right side of the liver from the abscesses of the left encircles the liver, and passes through the long axis of the gall-bladder and the long axis of that part of the inferior vena cava which passes through the liver (*Fig.* 482). This line would include in the left lobe a triangular area on the anterior surface of the liver, between the falciform ligament, and a line passing from the fundus of the gall-bladder to the point where the falciform ligament passes over the anterior to the superior surface of the liver. The line would place the quadrate and the caudate lobes in the left lobe of the liver.

The Right Lobe Abscesses.—

The Clinical Syndrome of Right Lobe Abscesses presenting below the Right Lower Chest Wall.—Pain and tenderness over the right lower intercostal spaces



A B

FIG. 482.—A, B, The line suggested for demarcating the right and left lobes of the liver.

A New Set of Clinical Syndromes characterizing Abscesses at Different Sites in the Liver.— A set of clinical syndromes based on symptoms and signs produced by the presentation of abscesses at different surfaces of the liver would determine not only that an abscess was in the liver, but also that the abscess was near a particular surface of the liver. Should the abscess rupture through the liver, the symptoms and signs would also be dependent on the lesion being at a particular site in the liver.

The clinical syndromes of abscesses in the liver, and of the complications consequent on the rupture of abscesses through the liver, are therefore best grouped according to the surfaces of the liver at which abscesses present themselves.

Right and Left Lobe Liver Abscesses.—Right lobe abscesses are far more frequent than left lobe abscesses. Most right lobe abscesses present under the right lower chest wall, causing pain and tenderness there, and this accounts for acceptance of these clinical findings as being the clinical findings of an abscess in the liver. Left lobe abscesses are latent till they produce a swelling in the epigastrium.

Right and left lobe abscesses of the liver are therefore separate clinical entities, and in this context the demarcation between right and left lobes is dependent not on the precise line demarcating liver formed from, and drained by, the right and left hepatic ducts, as on the configuration and surroundings of the right and left parts of the liver.

FIG. 483.—A large right lobe liver abscess. The patient is lying on the operating table ready for aspiration of a large liver abscess. The distension of the abdomen, the dilated veins on the lower chest and the abdominal wall, the bulging of the lower intercostal spaces, and the emaciation of the patient are depicted.

are so often met with in cases of abscess of the liver that they are regarded as the cardinal signs of a liver abscess. The tenderness is usually localized over a small area involving one or a few adjacent intercostal spaces, but it may involve the whole of the right lower chest wall. There may be bulging of the intercostal spaces and pitting œdema of the subcutaneous tissues (*Fig.* 483). This pain and tenderness are clearly indicative of an underlying inflammation, and the abscess in the liver must be beneath

this area. The abscess must therefore be presenting on the surface of the liver underlying the tender intercostal spaces. These must therefore be right lobe liver abscesses presenting on the surface of the liver underlying the chest wall. The question whether there be pus in every case with this clinical syndrome has been discussed in the section dealing with amœbic hepatitis.

Would an abscess in the right lobe of the liver cause pain before it reached the surface of the liver? If it did so there would be pain, but no tenderness over the right lower intercostal spaces, because the interposition of the diaphragm and the chest wall between the liver and an examining finger on the chest wall would allow detection of tenderness only when the abscess reached the surface of the liver. The records of cases show, however, that pain and tenderness are found together. Both the pain and the tenderness therefore occur at the same time—that is, when the abscess reaches the surface of the liver. Should such an abscess rupture on the surface of the liver, the pus leaking from the abscess would cause extension of suppuration beyond the liver. The rupture of a liver abscess on the surface of the right lobe under the chest wall usually causes a general peritonitis, with a pool of pus between the muscular part of the diaphragm and the liver at the site of the rupture. There is little tendency to form a subphrenic abscess between the liver and the diaphragm, probably because of the respiratory movements causing the liver to move down the muscular wall of the diaphragm, so preventing localization of pus between the diaphragm and the liver. For the same reason an abscess presenting on the surface of the liver under the chest wall does not erode its way through the muscular wall of the diaphragm to present on the chest wall, although it does cause sufficient reactionary inflammation of the chest wall to give swelling of intercostal spaces and pitting œdema of tissues.

The Amœbic Abscess which presents on the Superior Surface of the Right Lobe of the Liver.—How could an amœbic abscess presenting on the superior surface of the right lobe of the liver be detected? The abscess would lie immediately below the central tendon of the right cupola of the diaphragm, and the inflammation could extend to the under-surface of its central tendon. This would cause pain referred to the top of the right shoulder. In a tropical country, a patient with the general constitutional disturbances of a pyogenic inflammation, having pain on top of right shoulder, should be regarded as being possibly a liver abscess presenting on the superior surface of the right lobe. These abscesses are often so large that they often also present under the right chest wall, causing pain and tenderness over the right intercostal spaces, which gives a clear indication of the diagnosis. It is necessary to take immediate note of a case with pain on top of the right shoulder. Failure to do so would expose the patient to the hazard of rupture of the abscess through the top surface of the right lobe either into the peritoneal cavity, or, if adhesions have formed, either into the right pleural cavity or into the right lung. The hazard of rupture through the central tendon of the diaphragm contrasts with the rarity with which an abscess ruptures through the muscular walls of the diaphragm. This is probably due to the lack of movement on respiration between the central tendon of the diaphragm and the upper surface of the right lobe of the liver as contrasted with the wide excursion on respiration between the liver and the muscular wall of the diaphragm.

Case Report illustrating the Importance of Pain on Top of the Right Shoulder.—Mrs. N., aged 30 years, had been treated in April, 1931, for a liver abscess over seven days with daily injection of emetine hydrochloride gr. 1, when she developed pain on top of the right shoulder. She was also tender over the right lower intercostal spaces. She was advised that, despite the treatment she had had, the abscess was enlarging and was now presenting on the top of the right lobe of the liver. An immediate aspiration of pus from the abscess was advised to avert the danger of rupture of the abscess. The patient refused to allow this and discontinued treatment. Three days later she had a sudden agonizing pain in the right chest, with dyspnœa. She was profoundly shocked and died in a few hours. It is very probable that in her case the abscess had suddenly ruptured into the right pleural cavity, causing death from pleural shock.

The Rupture of a Liver Abscess through the Superior Surface of the Right Lobe of the Liver.—The rupture of a liver abscess through the superior surface of the right lobe of the liver is generally an unheralded event. The abscess is latent till it presents on the superior surface of the liver, and it will cause pain over the top of the right shoulder only if there be inflammation extending beyond the liver to the under-surface of the central tendon of the diaphragm. Should the abscess rupture into a clean peritoneal cavity, the first intimation of the abscess will be the onset of acute abdominal pain with board-like rigidity. The pain over the top of the right shoulder occurring at this time may be unnoticed on account of the greater severity of the pain in the abdomen. If the patient should delay a few days before coming for advice, he will be seen at the stage of a general peritonitis.

The abscess may erode a passage through the central tendon of the diaphragm, causing no evidence of this till the abscess ruptures either into a clean pleural cavity or into the right lung which has become adherent to the diaphragm. Rupture into the pleural cavity may result in an empyema, the pleural cavity being filled with chocolate or creamy glairy pus. Rupture into the right lung causes the abscess to work a passage through the lung into an inferior lobe bronchus, and the patient then coughs up pus which is typical of a liver abscess.

A Liver Abscess rupturing into the Right Pleural Cavity.—

Case Report.—H. V. S., male, Sinhalese, aged 32 years, was admitted to hospital on July 5, 1946, on account of pain in the right chest and breathlessness of two weeks' duration. Temperature 99·2°. Whole of right chest dull on percussion.

X-ray appearances were those of a right pleural effusion.

July 9: Chocolate pus aspirated from right chest.

July 12: Operation under local infiltration anæsthesia. The eighth rib was resected and chocolate pus drained from the right pleural cavity.

Aug. 4: The wound was reopened and chocolate pus drained again from the pleural cavity.

Oct. 6: Discharged from hospital fit and well.

A Liver Abscess rupturing into the Right Lung.—*Case Report.*—G. D., male, Indian Tamil, aged 33 years, was admitted to the General Hospital, Colombo, on Nov. 30, 1952, on account of fever with rigors of two weeks' duration. There was pain on top of the right shoulder from the onset and pain in the right upper abdomen of four days' duration, with coughing up of chocolate cream pus. Temperature 103·2°. X-ray opacity of right chest up to second interspace. Heart was not displaced.

Dec. 18: Œdema of right chest wall with tenderness up to the second right interspace. Chocolate cream pus was still coughed up. Needle inserted in ninth interspace and 50 c.c. of chocolate cream pus aspirated.

Dec. 23: Left hospital feeling fit and well.

Amœbic Abscesses presenting as Swellings in the Right Loin.—The amœbic abscesses presenting as swellings in the right loin are an interesting group, which can be clinically differentiated according to their positions in the liver.

An Abscess presenting on the Bare Area of the Liver.—The bare area of the liver is the only surface of the liver in contact with the extraperitoneal bed of the posterior abdominal wall. An abscess presenting on the bare area would not cause pain and tenderness over the right lower chest posteriorly, because of this area being deep to the right erector spinæ muscle mass. The abscess would be latent.

Should the abscess rupture through the liver at this place the pus will leak into the retroperitoneal bed and will track behind the right suprarenal gland and the right kidney to present as a painful swelling in the right loin. This swelling would be indistinguishable from a perinephric abscess, and the abscess would be opened and drained on a diagnosis of perinephric abscess. At operation the pus would be in the perinephric space, and the diagnosis of liver abscess would be suggested by the glairy chocolate or creamy pus which escapes.

Pyogenic micro-organisms causing true perinephric abscesses would also on occasion form glairy chocolate or creamy pus. The diagnosis of amœbic abscess could be established if there were amœbæ in the pus. It would be established if pus were withdrawn from the liver by aspiration through an exploring needle. In other cases the diagnosis would be in doubt. It would be best not to continue to drain an abscess cavity by a drainage tube in cases where glairy chocolate or creamy pus was found in a perinephric abscess.

An Abscess presenting at the Postero-inferior Corner of the Right Lobe.—The postero-inferior corner of the right lobe of the liver has a posterior, a lateral, and an inferior surface. The inferior surface is indented by the renal impression.

An abscess within the liver at this place would give a round tender tumour in the right loin moving on respiration and coming from under cover of the costal arch. The tumour would have the physical signs of an enlarged and painful right kidney. In these cases, after investigations have established that the tumour is not a kidney tumour, the lump is exposed at a surgical operation to determine its nature. The lump is seen to be within the liver, and an exploring needle will withdraw the viscid pus of an amœbic abscess.

Should an abscess at this site rupture through the liver, it will cause either a peritonitis or a localized intraperitoneal abscess in the right loin. A localized intraperitoneal abscess in the right loin simulates a perinephric abscess very closely, and a diagnosis is not made until the lesion is explored at an operation. The abscess will then be found to be intraperitoneal.

An Amœbic Abscess presenting as a Swelling in the Right Hypochondrium.—The right hypochondrium demarcated by the vertical lines and the horizontal planes defined by anatomists is a triangle of so limited an area as to be of little value for defining the position of swellings in the abdomen. In clinical work a swelling in the right hypochondrium would be a swelling in the right upper abdomen lateral to the outer border of the rectus abdominis muscle. An amœbic abscess presenting as a swelling in the right hypochondrium would emerge from under the right costal margin lateral to the rectus abdominis muscle. If the abscess be within the liver, the liver has enlarged to project below the right costal margin. The anterior surface of the right lobe now extends below the costal margin, and the inferior surface at the site of its colic impression is bulged downwards. The swelling moves on respiration, it is tender, and it is continuous with the liver. In tropical countries a swelling at this place would be recognized to be an amœbic abscess of the liver. A swelling at the same site might be an intraperitoneal pool of pus from rupture of an abscess of the liver either on its anterior surface lateral to the gall-bladder, or on its inferior surface over its colic impression. The intraperitoneal abscess would not move on respiration. In a tropical country the swelling would be recognized to be an intraperitoneal abscess from the rupture of a liver abscess.

Liver Abscesses presenting in the Right Loin.—

Case Report: A Liver Abscess rupturing into the Perinephric Space.—W. M. M., male, Sinhalese, aged 22 years, was admitted on Feb. 15, 1945 for pain and tenderness in the right loin of two weeks' duration. There was a soft fluctuant swelling bulging the right loin.

Operation on Feb. 19, under general anæsthesia. An incision 1 in. long was made in the right loin and chocolate-sauce pus drained from the perinephric space.

The patient made a good recovery and left hospital on April 21.

Case Report: An Abscess within the Right Lobe of the Liver presenting in the Right Loin.—A. K., male, Sinhalese, aged 14 years, was admitted on May 25, 1945 with pain in the right loin of three days' duration. He keeps the right hip flexed. On examination, temperature 103°; pulse 144; tenderness and fullness in the right loin; right flank is rigid and tender. A diagnosis of perinephric abscess was made.

Operation under spinal anæsthesia. An incision was made in the right loin. No pus in the perinephric space. A lump was felt in the liver at this place. A needle inserted into the liver aspirated foul pus with an offensive odour.

Case Report: A Liver Abscess which simulated an Enlarged Tender Kidney.—G. E. F., a male, Sinhalese, aged 45 years, was admitted on June 10, 1941 for pain in the right side of the abdomen of nine days' duration. The pain was accompanied by headaches and chills. The pain was diffuse at the commencement and after two days localized in the right lumbar region.

Temperature 99·4°. A round tumour was felt in the right loin emerging from under the costal margin and moving on respiration. A cystoscopy and direct pyelogram demonstrated that the pelvis and calices of the right kidney were normal.

Operation under spinal anæsthesia: The abdominal cavity was opened through a right paramedian incision. The round tumour was in the liver. A needle was inserted into the tumour and an ounce of chocolate-sauce pus was aspirated from the lump.

The patient made a good recovery from the operation and left hospital on July 5.

The Left Lobe Abscesses (*Fig.* 484).—

Epigastric Swellings from Amœbic Abscesses of the Liver.—An epigastric swelling is the second most common manifestation of a liver abscess. The swelling is due either to an abscess which is still wholly within the liver, or to an intraperitoneal abscess formed from pus which has leaked from a liver abscess. A careful evaluation of the physical signs of these cases permits these two types of abscess to be differentiated and also permits recognition of the precise site of each of these two types of abscess.

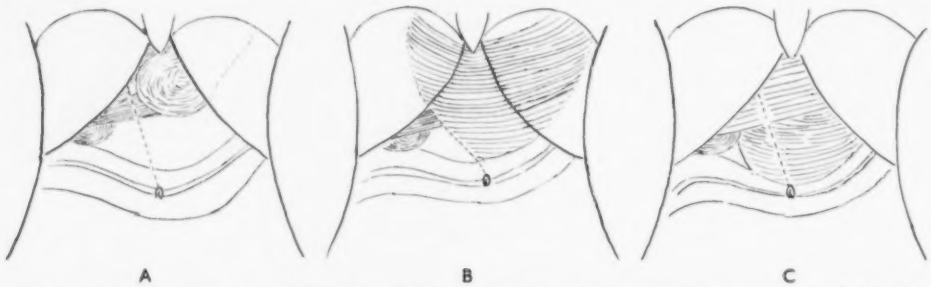


FIG. 484.—Epigastric abscess. A, An epigastric abscess within the liver. B, An epigastric abscess from the rupture of a liver abscess on the superior or anterior surfaces of the left lobe. C, An epigastric abscess from the rupture of an abscess on the inferior surface of the left lobe.

An Epigastric Swelling from an Abscess which is wholly within the Left Lobe of the Liver.—A liver of normal proportions is wholly under cover of the costal margins, except at the V-gap between the two costal arches, where the liver is immediately below the anterior abdominal wall. The edge of the liver at this place extends obliquely across the V-gap from the tip of the ninth right costal cartilage to the tip of the eighth left costal cartilage. An abscess in the liver at this place would cause an epigastric swelling in the upper recess of the V-gap. The swelling would be both painful and tender. The swelling is recognized to be wholly within the liver from the circumstance that it moves freely with respiration.

The epigastric swelling is deep to the linea alba and the left rectus abdominis muscle. The abscess is in the left lobe of the liver to the left of the falciform ligament. An abscess at this place would also bulge the inferior surface of the left lobe of the liver.

An abscess wholly within the liver at this place and causing a swelling in the epigastrium can be recognized with such certainty that a needle can be inserted into the abscess through the skin overlying the swelling, and the abscess emptied by aspiration of pus from it.

Intraperitoneal Abscesses from Rupture of Liver Abscesses causing Epigastric Swellings.—Intraperitoneal abscesses bulging the epigastrium lie immediately below the anterior abdominal wall. These swellings can be clearly differentiated from abscesses which are wholly within the liver from the circumstance that they do not move with respiration. Examination of the site of the swelling gives a clue to the source of the leak of pus from the liver.

If the pus was leaking from the anterior surface of the liver, the pus would collect between the anterior surface of the liver and the anterior abdominal wall. The pus extends upwards under the costal margins, and it is walled below by adhesions, gluing the greater omentum and the transverse colon to the anterior abdominal wall. The lower boundary of the abscess is palpable through the abdominal wall. Although the abscess extends upwards deep to the costal margins, it appears to be extending over the costal margin because of the rectus abdominis muscles tenting the swelling from their origins on the front of the costal cartilages. An abscess lying in front of the liver in the epigastrium is an intraperitoneal abscess from the leaking of an abscess on the anterior or superior surfaces of the left lobe of the liver. The pus would be subdiaphragmatic if the leak were from the superior surface of the left lobe or from that part of the anterior surface of the left lobe covered by the chest wall. The falciform ligament walls off these abscesses from the surface of the right lobe of the liver.

An intraperitoneal epigastric abscess from the leaking of an abscess on the inferior surface of the liver collects between the liver and the stomach in the first instance and, as the abscess enlarges, it reaches the deep surface of the anterior abdominal wall below the edge of the liver. The abscess is walled below by adhesions of the greater omentum and the transverse colon to the anterior abdominal wall, and this border is palpable through the anterior abdominal wall. Unlike the abscess derived from a leak on the anterior or superior surfaces of the left lobe, these abscesses bulge the anterior abdominal wall below the liver, which occupies the upper part of the V-gap between the costal arches. Owing to the falciform ligament not partitioning the space below the inferior surface of the liver, intraperitoneal abscesses below the liver could be from a leak of an abscess either through the inferior surface of the left lobe of the liver or through the quadrate of the lobe of the liver.

The epigastric swellings formed by intraperitoneal abscesses extend over a wider area than that demarcated as the epigastrium by the lateral lines and

horizontal planes of anatomists. There is good ground for abandoning anatomical definitions of the epigastrium for they do not conform to the natural boundaries met with in clinical work. The epigastrium is the upper middle part of the abdomen, and intraperitoneal epigastric abscesses demonstrate that the lower boundary of the epigastrium is a curved line convex down, passing from the tip of one ninth costal cartilage to the tip of the other, and with the curve passing across the umbilicus in the middle line.

How may an intraperitoneal epigastric abscess be treated? These abscesses can be clearly recognized and, once recognized, pus can be aspirated from these abscesses by a needle passed obliquely upwards into the abscess through the skin overlying the epigastric swelling.

An Intraperitoneal Epigastric Abscess from Rupture of a Liver Abscess on the Anterior or Superior Surfaces of the Left Lobe.—

Case Report: P. S., male, Sinhalese, aged 48 years, was admitted to hospital on Feb. 24, 1959 for a painful swelling in the epigastrium under the left rectus abdominis muscle. The swelling was of fifteen days' duration and there had been fever with chills every evening for the last thirteen days. In the last ten days there had been a constant pain on top of the left shoulder, which was not aggravated by taking a deep breath, although this caused pain in the epigastric lump. There had been an ineffective cough for the last ten days.

On examination the patient was thin and wiry. There was a tender swelling under the left rectus abdominis muscle reaching down from under the left costal margin, and with a well-defined lower margin bordered by palpable colon. It did not move on respiration.

A needle inserted into the swelling through the overlying skin withdrew 100 c.c. of creamy pus by aspiration. The patient left hospital on March 7, fit and well.

An Intraperitoneal Epigastric Abscess from Rupture of a Liver Abscess on the Inferior Surface of the Left Lobe.—

Case Report: B. A., male, Sinhalese, aged 57 years, was admitted on April 10, 1958 for a painful lump in the epigastrium of ten days' duration. He had had fever for the last fifteen days.

There was a disk-like swelling under the left rectus abdominis muscle well below the costal margin and with well-defined edges. It did not move on respiration. A needle was inserted into the lump through the overlying skin and 250 c.c. of creamy pus were aspirated. The patient left hospital on April 19 feeling fit and well.

Liver Abscesses simulating Acute Mucoceles or Empyemas of the Gall-bladder.—An abscess in the liver sometimes projects as a rounded tumour from the edge of the liver, below the right costal margin, at the outer edge of the rectus abdominis muscle. Such an abscess would present as a round tender tumour having all the physical signs of a mucocele or empyema of the gall-bladder. The abscess lies within the liver at the base of a triangle on the anterior surface of the liver between the falciform ligament and a line drawn from the fundus of the gall-bladder to the point on the anterior surface of the liver where the falciform ligament passes over to the superior surface of the liver. The abscess bulges the inferior surface of the liver, and here the bulge is in the quadrate lobe of the liver.

How is the problem of the diagnosis of this rounded lump dealt with? In many cases the possibility that the rounded lump is an abscess is overlooked, and the diagnosis is only made when the abdomen has been opened to deal with an acutely enlarged gall-bladder. The diagnosis of an abscess in the liver could be established by taking a cholecystogram, for an abscess in the liver would be

FIG. 485.—An amœbic abscess presenting as a rounded intra-abdominal lump simulating an empyema of the gall-bladder.

associated with a normal gall-bladder which would show a good shadow on the cholecystograms, while a mucocele or empyema of the gall-bladder would show no shadow of the gall-bladder in a cholecystogram.

Cholecystograms are not used, however, even though the diagnosis be in doubt, on account of the delay that such an examination would involve. In the series of cases under review a case with a lump which could have been either a gall-bladder or a liver abscess was taken up for operation, the abdomen was opened through a 2-in. incision, and the lump was inspected. If it was an abscess, a needle was inserted into it, and the abscess was aspirated of its pus. If it was an enlarged gall-bladder, the abdominal incision was enlarged and the gall-bladder removed.

Intraperitoneal Abscesses simulating Mucoceles or Empyemas of the Gall-bladder.—An intraperitoneal pool of pus could form a rounded tumour projecting below the right costal margin at the outer edge of the rectus abdominis muscle. An intraperitoneal

abscess at this place would be under the anterior abdominal wall and it could be differentiated from an abscess within the liver and from a mucocele or empyema of the gall-bladder from the circumstance that it would not move on respiration. Appreciation of this physical sign permits an abscess of this kind from being emptied of its pus by insertion of a needle into it through the overlying skin, and aspiration of the pus.

Amœbic Abscess presenting like an Enlarged Gall-bladder (*Fig.* 485).—

Case Report: P. A. W. S., male, Sinhalese, aged 22 years, admitted on Feb. 15, 1959, complained of a painful lump in the right upper abdomen of two weeks' duration. The pain had been noted first, and the lump on the next day. The pain was continuous, with exacerbations on taking a deep breath.

Three months ago, he had had loose stools, without blood or mucus, over a period of a month.

On examination the patient was emaciated and toxic. Temperature 98°. A globular lump the size of a billiard ball projected below the right costal margin beneath the outer border of the rectus abdominis muscle. The lump did not move on respiration. The liver was tender and enlarged a finger's breadth below the right costal margin. There was no tenderness over the right intercostal space.

The globular swelling was recognized to be a liver abscess adherent to the anterior abdominal wall. A needle was inserted into the lump through the overlying skin and 160 c.c. of creamy pus streaked with raspberry jam was removed by aspiration with a 50-c.c. syringe.

The patient made an uninterrupted recovery, and left hospital on March 6.

An Abscess in the Quadrate Lobe. The Syndrome is that of a Left Lobe Abscess (*Fig.* 486).—

Case Report: R. A. J. A., male, Sinhalese, aged 44 years, was admitted to hospital on Sept. 12, 1955 complaining of epigastric pain of three weeks' duration.

On examination a tender fluctuant mass was felt in the epigastrium.

Sept. 15: Operation under general anæsthesia. Abdomen opened through a right paramedian incision. There was an abscess extending from below the liver on to the front of the stomach. This abscess was emptied of pus. On raising the liver off the front of the abscess, a flaccid abscess cavity was seen occupying the whole of the quadrate lobe and with a rupture in its wall. The pus within this abscess was mopped out and the abdominal wound was closed. The patient made an uninterrupted recovery and left hospital on Sept. 28.

An Abscess in the Caudate Lobe of the Liver (*Fig.* 487).—What clinical syndrome could an abscess in the caudate lobe give? There was one case of an abscess in the caudate lobe in this series, and the evidence of this case established that an abscess in the caudate lobe is latent till it ruptures through the liver. The pus will leak into the lesser sac, and the intraperitoneal abscess behind the stomach is palpable as a tender mass in the epigastrium below the liver. The details of this syndrome would best be set forth by the report of this case.

Case Report: Abscess in the caudate lobe of the liver. R., Indian Tamil, male, aged 52 years, was admitted to hospital on Feb. 11, 1953 on account of continuous fever of two weeks' duration and pain in the epigastrium for the same period. The pain was noted before the fever. The patient was emaciated and toxic. There was a tender lump palpable in the epigastrium below the liver. The patient was considered to have a left-lobe liver abscess presenting in the epigastrium and he was put on daily injections of emetine hydrochloride, gr. 1.

On Feb. 23 an operation was performed under general anæsthesia. A right supra-umbilical paramedian incision was made. A lump the size of a fist was felt deep to the stomach. The inferior surface of the liver was fixed

FIG. 486.—An epigastric mass from rupture of an abscess through the quadrate lobe of the liver.

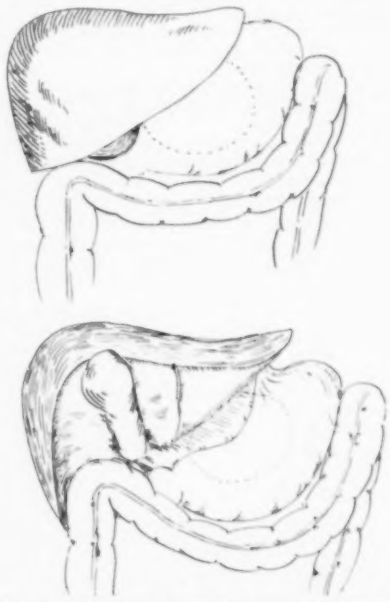


FIG. 487.—An epigastric mass from rupture of an abscess in the caudate lobe.

to the anterior surface of the stomach by filmy adhesions and on separating these and lifting up the liver, a round lump was found extending from the liver to behind the stomach. The lump was in the lesser sac. A needle was inserted into the lump through the lesser omentum and 60 c.c. of chocolate pus was aspirated from the lump, deflating it. The abdominal incision was closed without drainage. The patient made a good recovery from the operation and left hospital on March 4.

Subdiaphragmatic Abscess from the Rupture of a Liver Abscess.—An abscess in the right lobe of the liver rupturing under the right cupola of the diaphragm should on occasion form a subdiaphragmatic abscess. Pus collecting at this place would give the same physical signs as a right lobe abscess presenting under the right chest wall and under the central tendon of the diaphragm. Such a case would be treated by the insertion of a needle through the right lower intercostal spaces and aspiration of the pus. There would be no indication while this was being done that the pus was subdiaphragmatic and not within the liver, and these cases would not be recognized to be subdiaphragmatic abscesses from the rupture of a right lobe liver abscess.

A subdiaphragmatic pool of pus under the left cupola of the diaphragm would be from the rupture of an abscess on the superior or anterior surfaces of the left lobe of the liver. The abscess is latent till it extends below the left costal margin to form a swelling in the epigastrium. Such a case would be treated by aspiration of pus through a needle inserted into the swelling in the epigastrium. Whether the pus was subdiaphragmatic or whether it was localized to the epigastrium would be indicated by the amount of pus aspirated from the abscess. There might be tenderness over the lower left intercostal spaces.

Rupture of a Left Lobe Abscess into the Pericardium.—A left lobe abscess presenting under the central tendon of the diaphragm could rupture through this tendon into the pericardium. There were no cases of this dire complication in the present series of cases.

The Rupture of a Liver Abscess into the General Peritoneal Cavity.—The liver is enclosed by peritoneum on every surface except the bare area. A liver abscess reaching any surface of the liver but the bare area will rupture into the peritoneal cavity if adhesions have not formed to prevent this. At the onset the leak of pus into the peritoneal cavity causes acute abdominal pain and board-like rigidity of the abdominal wall. The picture is very much that of the perforation of a gastric or duodenal ulcer. Should the leak continue, the pus in the peritoneal cavity sets up peritonitis and toxæmia, and death will result. In the early stages of a leak from a liver abscess, the acute abdominal pain and the board-like rigidity will make it necessary to differentiate the condition from a perforated gastric or duodenal ulcer. An X-ray picture with the patient sitting up will show free gas under the cupolæ of the diaphragm in perforation of gastric or duodenal ulcers. In the liver abscess cases a hint as to the diagnosis may come from the concomitant presence of tenderness over the lower right intercostal spaces. In some of the cases where the abdomen was opened on a diagnosis of perforation of gastric or duodenal ulcer, and where a leaking liver abscess was found, the pus was observed to be exuding from the abscess like toothpaste from a tube. The rate of escape of pus was extremely slow, and aspiration of pus from the liver stopped the escape of pus from the abscess. As a result of these experiences, in subsequent cases where an early acute abdominal crisis was suspected to be a leaking liver abscess, a needle was inserted into the liver through one of the lower right intercostal spaces. If the diagnosis was established by the withdrawal of pus the abscess was then emptied of pus by aspiration through the needle. These patients made good recoveries, although the pus which had already leaked into the peritoneum was not removed. The method was used with success even when the pus was thin, provided that the aspiration was done early. The sudden onset of acute abdominal pain with board-like rigidity of the abdominal wall and with tenderness over the right lower intercostal spaces would be best managed in a tropical country by exploration of the liver by a needle inserted through the right chest wall. If pus is drawn, the abscess is emptied of pus by aspiration and recovery can be expected.

At the stage of peritonitis with a large volume of pus in the peritoneal cavity, mopping out of all the pus at a laparotomy occasionally determines recovery from an apparently hopeless condition. At such operations the site of the leak should be located, as the liver should be emptied of pus to prevent further leakage from the abscess. To do this the surfaces of the liver should be examined systematically. On the right lobe a leak occurs from the lateral, the superior, or the inferior surfaces. On the left lobe the leak could be from its anterior, superior, or inferior surfaces. The cases coming in at the stage of general peritonitis are usually not recognized as being from leaking liver abscesses. The abdomen is opened to deal with the cause of the general peritonitis. The absence of free gas in the peritoneal cavity and the presence of chocolate or creamy pus with a glairy consistence indicates that a liver abscess has ruptured into the general peritoneal cavity. The hand should be passed over the surfaces of the liver to locate the site of the rupture in the liver. Pooling of pus at the site of the rupture will direct attention to the site of the rupture in the liver, and an examining finger inserted at this place will detect the hole in the abscess wall. A guide to the site of the rupture sometimes comes from there being a localized pool of pus at the site of the rupture. This pool was not walled in sufficiently to prevent leakage into the general peritoneal cavity, and this occurs most often with abscesses rupturing through the inferior surface of the right lobe of the liver, or through the surfaces of the liver covered by the muscular part of the diaphragm.

General Peritonitis from the Rupture of Right Lobe Abscesses.—

Case Report.—P. P., male, Sinhalese, 45 years old, admitted on Feb. 11, 1954 for continuous fever of ten days' duration with chills and rigors. On examination, temperature 103°, pulse 102. Abdomen distended and rigid. Liver dullness present. A few crepitations at the right base. A diagnosis of perforation of a typhoid ulcer of the ileum was made and an operation under general anæsthesia was performed. The peritoneal cavity was opened through a McBurney incision and chocolate-sauce pus welled out. As much of this pus as could be got at was sucked out through this incision. A needle inserted

into the liver through the 8th right intercostal space aspirated chocolate-sauce pus from the liver. The McBurney incision was closed. The patient made a good recovery and left hospital on March 3.

Case Report.—P. L. L. P., male, Sinhalese, 36 years old, was admitted on Dec. 16, 1954 for acute hepatitis with right-sided pleurisy of a week's duration. He had had a bout of dysentery with blood and mucus in the stools a month earlier. On examination, the patient was groaning in agony and there was a tense tympanitic distension of the right lower abdomen. As the condition failed to improve on treatment with amœbicidal drugs an operation was performed on Dec. 18. The peritoneal cavity was opened through a right paramedian incision. Chocolate-sauce pus welled out from the under-surface of the right surface of the liver. On lifting up the right lobe, a rupture of a liver abscess was seen just above the right kidney. The pus was sucked out and the abdominal cavity was closed. The patient made a good recovery and left hospital on Dec. 30.

A Series of Cases to show the Relative Incidence and Mortality of the Different Clinical Syndromes

	Recoveries	*Deaths*
Amœbic hepatitis (cases with pain and tenderness of the right lobe of the liver, and in which pus was either not searched for or not found)	56	1
Right lobe abscesses in which pus was aspirated through the right chest	31	4
Right lobe abscesses eroding a passage through the right cupola of the diaphragm	13	4
Right lobe abscesses presenting at and drained through the right hypochondrium	12	1
Right lobe abscesses presenting at and drained through the right loin	6	0
Left lobe abscesses presenting in the epigastrium	40	3
Abscesses simulating an empyema of the gall-bladder	8	0
The rupture of a liver abscess causing general peritonitis	8	8
Death from multiple large abscesses of the liver	0	5
	174	26

PART III

The Evacuation of Pus from a Liver Abscess.—

The Open Drainage of a Liver Abscess.—The drainage of pus from a liver abscess was practised with success by Hippocrates. Drainage of pus has been replaced by aspiration of pus, because of the considerable delay in healing occurring when an abscess is drained, as against the rapid recovery which is the rule when an abscess is aspirated.

What is the reason for the delay in healing when an abscess is drained? The delay in healing is attributed to the occurrence of secondary infection with pyogenic organisms coming in from the surface along the track of the drainage tube. Is this belief correct? The pus in an unopened liver abscess may contain pyogenic micro-organisms and the aspiration of such pus gives as quick a recovery as does the aspiration of pus which contains no micro-organisms. It is evident that pyogenic organisms in a liver abscess do not delay healing when pus is aspirated. Why should micro-organisms in the wound track behave differently? It is likely that the delay in healing is from the operation of some other factor. What could this factor be?

Fig. 488.—Extending necrosis of the abdominal wall at the site of drainage of an epigastric abscess. Photograph taken the day before death three months after the drainage.

Drainage of a liver abscess is very occasionally marred by a fearsome complication when the orifice of the drainage track enlarges to become an enormous crater opening into a sloughing cavity, the cavity of the liver abscess (*Fig.* 488). The toxæmia is so severe that these cases nearly always terminate fatally. What has happened in these cases? The necrosis and the advancement of the granulations are due to invasion of the tissues by amœbæ, and these amœbæ have come to invade the tissues because the establishment of drainage permitted pus laden with amœbæ to flow over these tissues. This terrible complication gives a clue to a possible explanation for the delay in healing when a liver abscess is drained. Could this have favoured a lesser grade invasion of the wound track by amœbæ, delaying healing till these amœbæ are destroyed? That this is likely to be the case is seen by assessing the successes Hippocrates had with drainage of liver abscesses as against the poor results of drainage obtained since then, despite the advantage of the use of amœbicidal drugs. Hippocrates worked at a period long before the introduction of the drainage

tube. Simple drainage of pus as done by Hippocrates would be far less likely to allow amœbæ to infiltrate the drainage track than present-day drainage with a tube, which will be bathed in pus all the time that it is in place, with a further flow of pus over the wound track after the tube is removed.

These considerations dictate that an amœbic abscess of the liver should not be drained by a tube. This would apply even if amœbæ were absent from the pus, for the appearance of amœbæ in the pus at a later stage of drainage is quite common. Moreover, when an abscess of the liver is opened, pus should not be permitted to leak through the wound for several days after the operation. This could be prevented by emptying all pus from the abscess at the time that it is opened. Even if pus re-forms, the wound track will seal before pus forms again.

The Aspiration of Pus from a Liver Abscess.—The pioneers of the method of emptying an abscess of the liver by aspiration through a cannula inserted through the skin were impressed by the problem of evacuating the thick pus usually found in the liver. The trocars and cannulas of Manson, Rickman Godlee, Cantlie, and Rogers were of a wide bore to facilitate the evacuation of thick pus. The trocars were so thick that they sometimes caused a fatal hæmorrhage from the liver. Surgeons working in tropical countries observed that although thick pus gushed out of the cannula, when the trocar was withdrawn the flow would cease before the abscess was empty, and using a wider-bore cannula did not improve the situation.

Much more pus could be withdrawn by suction, and when suction was employed it was observed that thick pus flows as well through a small-bore cannula as through a wider-bore cannula. The apparatus devised by Potain for the aspiration of pleural effusions came to be used widely for the aspiration of liver abscesses, and the small-bore cannulas of Potain, which were smaller than a slate pencil in diameter, proved to be as practicable for aspirating pus from a liver abscess as the wider-bore cannulas of the pioneers of aspiration, which were of the diameter of a pencil. Liver abscess pus flows through a small-bore cannula because of its having the viscosity of a thick lubricating oil. On occasion an abscess is filled with a solid jelly which will not flow through any cannula no matter how wide, but such lesions are rare. They cannot be evacuated by aspiration through a cannula.

Even with suction, the flow of pus from the abscess ceases before all pus has been emptied from the abscess. When the cannula is first inserted into the abscess, the pus escapes forcibly because of its being confined under pressure within its walls. The flow of pus ceases as soon as the pressure drops to atmospheric pressure. Suction with a hand pump can exert a negative pressure of 1 atmosphere and some electric pumps can exert a greater negative pressure than this, but more pus will flow from the abscess only if the wall of the abscess can collapse, and the wall of a liver abscess cannot collapse much. Air introduced into the abscess cavity would permit complete evacuation of pus from the abscess, but this method carries the risk of air embolism. Moreover, healing of an abscess occurs when an abscess is emptied of as much pus as can be aspirated from its cavity even though there must be residual pus in the abscess cavity. There is no risk of a leak of this residual pus from an abscess which has been as completely emptied of pus by aspiration as is possible, as the residual pus is not retained under pressure. The needle track will seal before sufficient pus can re-form to raise pressures in the abscess cavity.

What is the Best Method for aspirating Pus from a Liver Abscess? The exploration of a liver by a needle of 1–2 mm. in diameter is a preliminary to the aspiration of pus. Thick pus flows out even through so fine a needle, as the pus is under pressure within its walls. If a 500-c.c. or 100-c.c. syringe be connected to the needle, pus can be aspirated from the abscess even though it be thick. The syringe exerts a greater negative force than is possible with a hand pump, and moreover this force can be regulated according to the resistance to the flow of pus from the abscess. Aspiration of pus with a needle and syringe will empty a liver abscess of as much pus as can be got by aspiration. The small bore of the needle minimizes the risk of hæmorrhage from the track of the needle, and the consistence of liver abscess pus permits thick pus being aspirated through a needle of this small bore. The apparatus is compact and readily transportable, and the method has now been used in our clinic for several years with satisfaction.

How much Pus could a Liver Abscess contain?—The size of a liver abscess can be estimated from the volume of pus withdrawn from an abscess at a single sitting. The aspiration of ½–2 pints from a liver abscess is a common experience in tropical countries. In the present series of cases the maximum amount of pus withdrawn from a liver abscess at a single sitting was 4¾ pints. Waring (1854) recorded two cases in the East Indies in which the abscess contained 8 pints of pus. MacLean (1871) recorded a case containing 19 pints of pus, and Toman, of Liverpool, recorded 18 litres of pus from an abscess.

How does a liver abscess come to contain such large volumes of pus? The volume of a liver of a normal adult is just over 2 pints. It is clear that the liver must be expanded and distorted if it has to accommodate over a pint of pus. This is mainly done by elevation of the right cupola of the diaphragm to house the abscess within the right thoracic cage. The pressure of the pus in these large abscesses is great, and emptying the abscess of pus gives dramatic relief from the effects of these pressures.

The following report illustrates the rapid subsidence of pressure effects after a single aspiration of a liver abscess.

A male, aged 27 years, was admitted on Jan. 23, 1958 as a case of cirrhosis of the liver. The patient was emaciated and toxic and there was pitting œdema of both feet and legs and of the back. The skin and conjunctiva had an icteric tint. There was moderate abdominal distension, with large subcutaneous veins on the front of the trunk. There was marked tenderness over the right lower intercostal spaces. Jan. 24: Two and a half pints of chocolate pus were aspirated from the liver through the 9th right intercostal space. A course of amœbicidal drug therapy was given. On Feb. 1 there was no œdema and no dilated veins on the trunk. The icteric tint was barely perceptible. When seen on March 25 the patient presented the appearance of normal health.

The presence of over ½ oz. of pus in a liver is diagnostic of an amœbic abscess. An ounce of pus could not be removed by aspiration from the dilated channels of a suppurative pylephlebitis or a suppurative cholangitis.

Case Report: A Giant Abscess cured by a Single Aspiration of Pus.—P. S., a male Indian Tamil, aged 38 years, was admitted to the General Hospital, Colombo, as a case of advanced carcinoma of the stomach. He had a gross swelling of the upper abdomen, he was emaciated, and he had been in a district hospital for one month. The patient stated that at the onset of the illness five weeks previously, he had had fever with chills and rigors for three days but he had been afebrile since then. At the onset he had slight pain in the right lower chest, and this pain had persisted and was now much worse. The pain was aggravated when he breathed, and when he lay on the right side. He had also developed an upper abdominal swelling. On examination the patient was noted to be thin and wiry, but he did not look toxic. Temperature 98°. There was a gross distension of the upper abdomen reaching 1 in. below the right costal margin, and extending across to the left, where it was 3½ in. below the left costal margin. The whole of the epigastrium was bulged by the swelling and the swelling also bulged the right loin. The edge of the liver formed the margin of the swelling on the right, and here the edge moved downwards on respiration. The swelling was dull on percussion. The skin over the swelling was stretched and shiny and there were large dilated veins under the skin. The right chest did not move on respiration. The right intercostal spaces bulged outwards and there was dullness on percussion from the third intercostal space downwards. There was no œdema of the lower limbs. No shifting dullness in the abdomen. No jaundice, tint of eyes, or conjunctiva.

A diagnosis of a large liver abscess was made and aspiration was advised. The patient consented to have this done on March 27, 1958. Under local infiltration anæsthesia a needle inserted into the epigastric swelling drew no pus. A needle inserted through the 9th right intercostal space drew pus which was like coffee and milk at first and then became thick and like chocolate sauce; 90 oz. of pus were aspirated. The swelling in the epigastrium deflated completely with the aspiration, but the swelling of the right loin remained partially deflated. The patient was completely relieved by this aspiration. He received six injections of emetine hydrochloride 2/3 gr. and left hospital on April 11 to resume work as an estate labourer. The stools examined on April 10 showed *Entamœba histolytica* cysts.

The Plan of Treatment.—As a result of experience gained over a period of years, a plan of treatment has been evolved which has proved to have many advantages. Every case of amœbic abscess of the liver is admitted to hospital and put on a course of amœbicidal drugs, usually for 12 days. A needle is inserted into the liver and all the pus that can be aspirated is removed at one sitting. The needle is inserted at a site determined by a review of the clinical findings. In cases in which there is only a mild disturbance of the general health, needling is not done. A second insertion of the needle into the liver is done only if there is a recurrence of the symptoms and signs of toxæmia. This technique has proved successful even in cases with several pints of pus in a liver abscess, which have made a complete recovery after a single aspiration of pus from the abscess.

Acknowledgements.—The post-mortems on liver abscess cases in this series in 1957 and 1958 were done by Dr. N. Nagaratnam, to whom I am grateful for his meticulous demonstrations by histological examinations that, even in cases of multiple abscesses in the liver, the liver tissue beyond the walls of the abscesses was normal. These findings are in agreement with those of Rogers (1922) on amœbic abscesses, but they are contrary to observations of Councilman and Lafleur (1891), who describe a widespread necrosis of the cells around the central veins throughout the liver. I am grateful to Dr. Malinga Fernando, Research Assistant, Department of Surgery, for sharing so enthusiastically in the work for this paper. To Professor W. A. E. Karunaratne, Emeritus Professor of Pathology, and Dr. W. D. Ratnavale, Pathologist, General Hospital, Colombo, I make grateful acknowledgement of wise guidance in the conduct of the investigations. The diagrams were prepared by Mr. A. S. Mahawatte. The photographs were taken by Mr. P. D. S. Amerasekera and Mr. A. S. Mahawatte, of the Department of Photography, University of Ceylon, and by Mr. M. Jayasinghe, Photographer to the General Hospital, Colombo.

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HIATUS HERNIA: A CAUSE OF GASTRO-INTESTINAL HÆMORRHAGE

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As long ago as 1610 Ambroise Paré described a case of gastric herniation through the œsophageal hiatus (Bawditch, quoted by Ohler and Ritvo, 1943) and in 1836 Richard Bright gave an account of a "remarkable displacement of the stomach" associated with gastric ulceration. Only comparatively recently was hæmorrhage recognized as a complication of hiatus hernia. Ohnell described a case in 1926, and this report was followed by others from Bock (1929), Harrington (1929), and Truesdale (1932). Harrington, at that time with a total experience of 30 hiatus hernia operations, described 8 cases which had bled preoperatively. In 1933 Gardner reviewed 22 published cases of hæmorrhage associated with diaphragmatic herniation (probably not all hiatus hernias) to which he added 6 of his own. Sahler and Hampton (1943) noted that hæmorrhage usually complicated 'large' hernias, and Belsey (1954) stated that massive hæmorrhages were far more common with rolling hernias than with sliding ones. Lyons and Parodny (1956) confirmed this view; in their series, 60 per cent of para-œsophageal hernias compared with 13·3 per cent of sliding hernias were complicated by hæmorrhage. All these authors recognized that bleeding could arise from ulceration of the mucosa of the herniated portion of the stomach.

Table I.—Analysis of 138 Cases according to Sex and Type of Hernia

	Sliding	Rolling	Total
Male	49 (47 per cent)	6 (22 per cent)	55 (40 per cent)
Female	55 (53 per cent)	28 (78 per cent)	83 (60 per cent)
Total	104 (76 per cent)	34 (24 per cent)	138

In 1936 Vinson and Butt described 213 cases of œsophagitis out of 3032 necropsies of which 15 had been complicated by hæmatemesis or melæna. They did not mention hiatus hernia as a cause of œsophagitis and it remained for Dick and Hurst (1942) to emphasize this association. The latter also described anæmia and massive hæmorrhage as a complication of œsophagitis. Allison (1948) analysed 74 cases of hiatus hernia with œsophagitis, of which 10 bled seriously and 1 slightly. He later agreed with Barrett (1950) that reflux œsophagitis is not a cause of massive hæmorrhage, and that an œsophageal ulcer which does bleed seriously is usually a true gastric ulcer within an œsophagus lined by gastric mucosa (Allison and Johnstone, 1953).

Dick and Hurst also drew attention to the common accompaniment of reflux œsophagitis with gastro-duodenal ulceration, an observation which has been confirmed by Ohler and Ritvo (1943), Allison (1948), Winkelstein (1956), and many others. Wangensteen and Leven (1949) were so impressed by this association that they advised partial gastrectomy as a treatment for severe peptic œsophagitis.

From these observations it would appear that a case of hiatus hernia may bleed as a result of œsophagitis, duodenal ulceration, or gastric ulceration within the hernia itself. The complication is well documented and not at all uncommon. Ohler and Ritvo report 29 mild and 12 severe hæmorrhages (39 per cent) in a series of 104 hiatus hernias. Sahler and Hampton (1943) analysed 221 cases and found that 32 (15 per cent) had bled. Sweet (1952) describes 15 massive hæmorrhages in 111 consecutive cases of hiatus hernia (13·5 per cent). The objects of this paper are to reaffirm that hæmorrhage may complicate hiatus hernia; to determine its cause in large rolling, but otherwise uncomplicated, hernias; and to emphasize that this diagnosis should always be considered in cases of uninvestigated gastro-intestinal hæmorrhage.

Table II.—Analysis of Cases according to Severity of Hæmhorrage and Type of Hernia

	Total	Massive Hæmorrhage (Melæna or Hæmatemesis or Both)	Mild Hæmorrhage	Primarily Anæmia
Rolling hernia	15	10	1	4
Sliding hernia with œsophagitis	10	0	7	3
Incarcerated sliding hernia	3	2	1	0
Hiatus hernia with duodenal ulcer or other cause of hæmorrhage	9	7	2	0

Series under Consideration.—The cases here analysed are from a consecutive series of 138 adults with hiatus hernias. Children are excluded because it is believed that they comprise a separate pathological and clinical entity (Marchand, 1958). Four traction hernias due to fibrosis of the œsophagus (1 caustic, 2 scleroderma, and 1 following prolonged intubation) are also excluded. Fifty-five patients were men and 83 women. Forty-nine men and 55 women had sliding hernias; 6 men and 28 women had rolling hernias (*Table I*). On pathological grounds it is maintained that rolling hernias are not sub-divisible into para-œsophageal and composite hernias, and in

my experience upward displacement of the cardia can always be demonstrated (Marchand, 1958).

Thirty-seven patients (27 per cent) have bled at some stage of the disease (*Table II*). Fifteen of these had rolling hernias (44 per cent of the total rolling hernias) and 22 had sliding hernias (20 per cent of total sliding hernias). Hæmorrhage has been listed as massive when melæna or hæmatemesis has necessitated treatment by blood transfusion, and as mild when blood-staining of vomitus or the passage of tarry stools have not caused anæmia. In most 'mild' cases the presence of intestinal bleeding has been confirmed by a positive orthotolidine and amidopyrine reaction of the stools. Cases have been categorized as 'anæmia' when the patient has been admitted to hospital because of anæmia and not because of overt hæmorrhage, even though the history may have disclosed the occurrence of dark stools previously disregarded by the patient. All these cases have had a hæmoglobin level of less than 12 g. per cent (lowest level of normal at Johannesburg, altitude 6000 ft., has been taken as 13 g. per cent for women, 14 g. per cent for men).

Table III.—Analysis of Cases presenting with Massive Hæmorrhage

Case	Age (Years)	Sex	Form of Hæmorrhage	Length of History of Hæmorrhage	Type of Hernia	Incarceration	Œsophagitis	Cause of Hæmorrhage	Treatment	Result	Follow-up (Months)
1	80	F.	H. and M.	14 yr.	R.	+	0	H.G.U.	W.R. and I.H.	Died sixth d.	
2	58	F.	H.	9 yr.	R.	+	0	H.G.U.	D.H.	Recurrence	55
3	58	F.	H. and M.	First attack	R.	+	0	H.G.U.	I.H. and E.P.G.	Cured	38
4	62	M.	H. and M.	2 yr.	R.	+	0	A.U.	D.H.	Cured	31
5	59	M.	H. and M.	2½ yr.	R.	+	0	A.U.	D.H.	Cured	12
6	52	F.	M.	1 yr.	R.	+	0	A.U.	D.H.	Cured	11
7	76	F.	M.	4 yr.	R.	0	0	A.U.	D.H.	Cured	10
8	42	F.	H. and M.	First attack	R.	0	0	A.U.	D.H.	Cured	18
9	58	M.	M.	21 yr.	R.	0	0	A.U.	D.H.	Cured	24
10	70	F.	H. and M.	First attack	R.	0	0	A.U.	I.H.	Cured	42
11	58	M.	M.	3 yr.	S.	+	0	A.U.	D.H.	Cured	43
12	68	F.	H.	13 yr.	S.	+	+	A.U.	D.H.	Cured*	14
13	65	M.	M.	21 yr.	S.	0	0	O.V.	C.	Died 18 mth. later	—
14	42	M.	H.	First attack	S.	0	+	D.U. and G.U.	E.P.G.	Cured	48
15	43	M.	H. and M.	1 yr.	S.	0	+	D.U.	E.P.G.	Cured	45
16	46	M.	H. and M.	First attack	S.	0	+	D.U.	E.P.G.	Cured	26
17	75	M.	H.	First attack	S.	0	+	D.U.	C.	Died	—
18	50	F.	H. and M.	25 yr.	S.	0	+	G.U.	C. and D.H.	Cured	14
19	48	M.	H. and M.	3 yr.	S.	0	+	D.U.	E.P.G.	Cured	24

M., Melæna. H., Hæmatemesis. R., Rolling hernia. S., Sliding hernia. D.U., Duodenal ulcer. G.U., Gastric ulcer (outside of herniated stomach). H.G.U., Ulcer within herniated stomach. A.U., Acute ulcer or gastritis within herniated portion of stomach. O.V., Œsophageal varices. I.H., Immediate herniorrhaphy. D.H., Delayed herniorrhapy. E.P.G., Emergency partial gastrectomy. W.R., Wedge resection ulcer. C., Conservative.

MASSIVE HÆMORRHAGE

Nineteen patients were admitted to hospital with massive overt gastro-intestinal bleeding (*Table III*). Five experienced melæna alone, 4 hæmatemesis alone, and 10 both hæmatemesis and melæna. The cases are divisible into three groups:—

1. Hæmorrhage associated with rolling hernias—10 cases.
2. Hæmorrhage associated with incarcerated sliding hernias—2 cases.
3. Hæmorrhage due to causes unrelated to the hernia—7 cases.

1. Hæmorrhage associated with Rolling Hernias.—There were 3 men and 7 women in this group. The clinical details are presented by 3 illustrative cases.

Case 1.—Mrs. D. B., aged 80 years. Admitted June 12, 1958. Weight 122 lb., height 5 ft. 8½ in. This aged woman, mother of 3 children, experienced severe flatulent dyspepsia during and for some years after her pregnancies. At that time she weighed approximately 200 lb. Fourteen years previously she was admitted to hospital for the first time with hæmatemesis and had several blood transfusions. Thereafter she kept well though subject to high abdominal pain and left chest discomfort after meals, particularly if she went early to bed. In 1954 she felt faint and weak and noticed that her stools were black. About the same time she began to vomit freshly ingested food and this was associated with upper abdominal colic and dysphagia. At frequent intervals during the next 4 years she experienced attacks of abdominal colic and vomiting and

her weight dropped to 122 lb. On many occasions her stools were black. In March, 1958, she was admitted to hospital with hæmatemesis and melæna. Her hæmoglobin was 8·5 g. per cent. While in hospital she experienced frequent attacks of vomiting and abdominal colic and was obviously suffering from intermittent gastric obstruction. Radiography of the chest showed the presence of a fluid level within a lower mediastinal shadow and a barium meal demonstrated an incarcerated rolling hiatus hernia. Barium was retained within the stomach above the diaphragm for a considerable period and only trickled slowly into the abdominal stomach. A large ulcer crater was visible in the herniated stomach (*Fig.* 489). Operation was preceded by 5 days' preparation with blood transfusion and intragastric drip feeds.

FIG. 489.—Gastric ulcer in rolling hernia pouch situated immediately above constricting hiatal limbs.

AT OPERATION (June 16, 1958).—The chest and upper abdomen were opened through a thoraco-abdominal incision. The lung was densely adherent to the mediastinum. No ulcer crater was felt in the duodenum or sub-diaphragmatic stomach. The anterior surface of the stomach had rolled upwards through the large round hiatus into the hernial sac in front of the œsophagus. Disimpaction of the stomach was difficult because of gross thickening of its walls and dense perigastric mediastinal adhesions. With the stomach reduced it was noted that the induration involved about 3 in. of the lesser curve and anterior surface of the stomach immediately below the œsophagogastric junction, but did not reach the greater curvature. The gastro-hepatic omentum and left gastric artery were divided and the ulcer excised by wedge resection of the stomach. The excision encroached upon the cardia, but a sufficient lumen remained after repair. The hiatus was reconstituted and was reinforced with nylon mesh. The excised segment contained a deep punched-out gastric ulcer the size of a shilling into which an eroded vessel projected (*Fig.* 490). The patient progressed satisfactorily until the sixth day when she suddenly collapsed and died. No necropsy was done.

This case is representative of *Cases* 1, 2, and 3; all had incarcerated rolling hernias and chronic gastric ulcers within herniated stomachs. The ulcers constantly occurred on or close to the lesser curve immediately below the œsophagogastric junction, although as the stomachs lay in the mediastinum they were situated just above the level of the constricting hiatal edges. *Cases* 2 and 3 were œsophagoscoped and no œsophagitis was found. *Case* 3 was subjected to laparotomy undiagnosed because of uncontrollable hæmorrhage when an impacted hernia with a para-cardial ulcer was found. A high Polya gastrectomy was performed and the hernia repaired. The patient has remained well for four and a half years. *Case* 2 was operated upon during a remission through a thoracic approach and an aorta-œsophageal hernia (Adams and Lobb, 1954) was found. A para-cardial ulcer was palpated but not excised in the belief that it would heal once the hernia was reduced. Recovery was uneventful, but although the patient remained reasonably free from symptoms for about two years, the hernia was shown to have recurred within six months of operation. The hernia gradually increased to its pre-operative size, and thirty-two months later the patient again passed melæna stools. She has refused re-operation.

FIG. 490.—Part of stomach removed by wedge resection in *Case* 1. A deep punched-out chronic gastric ulcer is present.

Case 4.—Mr. S. P., aged 62 years. Admitted to thoracic surgical unit on June 24, 1956. Weight 185 lb., height 5 ft. 7½ in. This patient had never complained of dyspepsia. Two years previously he passed melæna stools and was admitted to hospital and transfused with 5 pints of blood. Within 2 days the hæmorrhage had ceased. Ten days later barium meal demonstrated an incarcerated rolling hernia, but no ulcer was seen in the stomach or duodenum. He declined further investigation and was discharged. From time to time he attended medical out-patients and remained well on treatment until Aug. 28, 1956, when he again passed massive melæna stools. With blood transfusions active bleeding soon ceased and a later barium examination confirmed the presence of the hernia (*Fig.* 491). Occult blood was found in his stools. A histamine gastric analysis showed a free acid level of 15 and total acid of 28.

FIG. 491.—Lateral barium radiograph showing a large rolling hiatus hernia which was the site of repeated gastric hæmorrhage. The stomach has undergone marked volvulus and a large pouch lies within a hernial sac in front of the œsophagus. No obvious ulceration can be seen.

AT OPERATION (Aug. 30, 1956).—Œsophagoscopy was done pre-operatively. No œsophagitis was seen. The stomach was entered 39 cm. from the incisor teeth and the cardia retracted on withdrawal to 34 cm. The chest was entered through the bed of the unresected eighth rib. A large hernia was present in the mediastinum. The diaphragm was opened widely and the stomach and duodenum were found to be free of ulceration. The hernia contained a large part of the anterior stomach wall below the cardia and about 4 in. of the lesser curvature. A clear line of transition demarcated the thickened wall of the herniated pouch from the rest of the stomach, but no palpable ulcer was present. A standard Allison repair was done. Recovery was uninterrupted.

The patient was seen on Nov. 17, 1958. He felt well, had, on instruction, lost 10 lb. in weight, and had not bled again.

This case represents *Cases* 4, 5, and 6 in that an incarcerated rolling hernia was present without œsophagitis or gastroduodenal ulceration. The whole wall of the herniated portion of the stomach was thickened and hyperæmic, but no discrete ulceration was found. *Cases* 4 and 5 were repaired through a thoracic approach and *Case* 6 through a thoraco-abdominal incision. There has been no further hæmorrhage 31, 12, and 11 months after operation.

FIG. 492.—Radiograph of upper abdomen two hours after barium meal (the patient remained recumbent throughout). Note barium retained within the herniated pouch of stomach.

Case 7.—Mrs. M. O., aged 76 years. Admitted March 31, 1958. Weight 139 lb., height 5 ft. 2 in. The patient, a childless widow, had suffered from severe dyspepsia until the age of 55 years. At that time she weighed 186 lb. Symptoms had improved after intentional weight-loss to below 150 lb., and until 1954 she had been well. In that year she experienced episodes of pallor, weakness, and dizziness and her stools were often black in colour. Similar attacks recurred many times, but only in March, 1958, was she admitted to hospital because of melæna. Hæmoglobin was 6·5 g. per cent, and she was transfused with 6 pints of blood over the course of a week. Barium examination revealed a rolling hiatus hernia, but no gastroduodenal ulceration was demonstrated. Histamine test meal was normal. A further barium meal showed that the hernia was reducible in the erect position, but after 2 hours' continued recumbency the herniated pouch of stomach remained outlined (*Fig.* 492).

AT OPERATION (March 14, 1958).—No œsophagitis was seen at œsophagoscopy. The chest and upper abdomen were opened through a thoraco-abdominal incision. A large hernia occupied the lower mediastinum. The cardia was displaced upwards and there was pouching of the stomach both in front and behind the œsophagus. No gastroduodenal ulceration could be felt. The hernia was easily reducible and no abnormality of the herniated stomach was detected from the serosal aspect. An Allison type of repair was reinforced by fixing a nylon mesh around the œsophagus on the thoracic aspect.

This case is representative of *Cases* 7, 8, 9, and 10. All had rolling hernias which reduced in the erect posture. With 3 patients, X-rayed after continued recumbency, the barium was still present in the herniated stomach several hours after the swallow. In no case was œsophagitis or gastroduodenal ulceration found radiologically or on direct palpation. At operation the hernias were easily reducible and no gross gastric abnormalities were detectable. *Cases* 8 and 9 were treated through thoracic incisions by the method of Allison. *Case* 10 was undiagnosed before surgery, and laparotomy was undertaken for persistent hæmorrhage. In the absence of duodenal or gastric ulcers the diaphragm was explored and the hernia found. The hiatus was repaired from below, and vagotomy and gastro-enterostomy were performed.

These 4 patients have remained free from hæmorrhage 10, 18, 24, and 42 months after operation.

2. Hæmorrhage associated with Incarcerated Sliding Hernias.—Two patients (*Cases* 11 and 12), a man of 58 years and a woman of 68 years, were admitted to hospital with severe anæmia (hæmoglobin level of 4 and 8 g. per cent respectively) after repeated attacks of melæna. Both had experienced dyspepsia for many years.

Case 11.—This man had previously undergone an abdominal operation at another hospital for repair of a hiatus hernia. Dyspeptic symptoms recurred within a few months and shortly afterwards the patient passed melæna stools for the first time. This persisted on and off until admission in August, 1955. Routine chest radiographs showed a rounded lower mediastinal shadow which, on barium examination, proved to be an incarcerated sliding hernia (*Fig.* 493). Barium remained within the hernia as long as the subject lay recumbent. No duodenal ulcer was demonstrable. No œsophagitis was seen at œsophagoscopy. Fractional test meal, using histamine, showed a total acid of 65 and a free acid of 35. Hæmoglobin was 4·6 g. per cent. After a period of treatment with blood replacement he was subjected to operation.

At Operation (Aug. 25, 1955).—Lateral thoracotomy, entry through the bed of the unresected seventh rib. The lung was adherent to the lower mediastinum. A large fixed sliding hernia was present. Adhesions were freed and the left vagus nerve had to be divided before the cardia could be placed below the diaphragm. The herniated stomach was thickened and hyperæmic, but no discrete ulceration could be felt. The bogginess of the stomach above the diaphragm was sharply demarcated by the edges of the hiatus, while below the stomach wall appeared normal. The diaphragm was opened and a standard Allison repair was done. Recovery was uneventful. When last seen the patient was well and asymptomatic. He had gained 18 lb. in weight, notwithstanding contrary advice, and had not bled since operation. Barium meal showed no sign of recurrence of the hernia.

Case 12.—This woman had had a cholecystectomy with removal of stones 8 years previously without relief. For 3 years she had suffered from dysphagia when eating hurriedly. Œsophagoscopy showed severe œsophagitis and stricture formation. Barium meal demonstrated a fixed hiatus hernia and the barium remained within the stomach pouch for several hours with the patient recumbent. No gastroduodenal ulcers were shown. The fractional test meal (histamine) showed normal acid values. At operation, through a thoraco-abdominal incision, no duodenal ulceration could be found. Peri-œsophageal adhesions were dense, but after extensive mobilization of the œsophagus, the cardia could be comfortably placed below the hiatus. No discrete ulcer was felt in the thickened and hyperæmic wall of the herniated gastric pouch. An Allison repair reinforced with nylon mesh was done. The patient has remained well for 14 months and a recent œsophagoscopy shows that the œsophagitis has cleared leaving minimal œsophageal narrowing.

Fig. 493.—Large incarcerated sliding hiatus hernia associated with hæmorrhage which arose within the gastric pouch.

3. Hæmorrhage unrelated to the Hernia.—Seven patients experienced massive gastro-intestinal hæmorrhage due to causes unrelated to the hernia. In 3 (*Cases* 14, 15, and 16), emergency laparotomies were done for persistent hæmorrhage, duodenal ulcers were found, and partial gastrectomies were performed. Hæmorrhage was cured, but dyspepsia continued, and post-operative barium studies demonstrated sliding hernias. With *Case* 13 a hiatus herniorrhaphy was done because of severe dyspepsia and œsophagitis, but eighteen months later the patient died from a massive bowel hæmorrhage. At necropsy a penetrating bleeding duodenal ulcer was found. *Case* 19 had a gastrectomy performed at an emergency operation despite the fact that no gastric or duodenal ulcer could be felt. The resected specimen was seen to be healthy when examined later. There is no note of an exploration of the hiatus during that operation. No further bleeding occurred, but because of dyspepsia a barium-meal examination was done and a hiatus hernia was demonstrated. We operated upon this patient six months later when a sliding hernia with a large peritoneal sac was found. It is possible that originally a rolling hernia was reduced by the operative manipulations and that full recurrence of the roll was prevented by the tethering effect of the gastrojejunal anastomosis. *Case* 18

had a single massive hæmatemesis and melæna eighteen months before admission to our wards. During the previous admission a pre-pyloric gastric ulcer had been shown and this had disappeared under conservative treatment. However, dyspepsia persisted and was later complicated by dysphagia. Severe œsophagitis with early stricture formation was present and an Allison repair of the hernia was performed. At operation no ulceration of the stomach or duodenum was felt, the hernia was reducible, and the herniated stomach looked normal. There was no evidence to incriminate the hernia as the source of the earlier hæmorrhage. Finally, *Case* 17 had a hiatus hernia complicated by gross œsophageal varices. No surgical treatment was undertaken and the patient died from hæmorrhage. Necropsy confirmed the absence of gastro-duodenal ulceration.

FIG. 494.—Large incarcerated rolling hiatus hernia associated with severe hypochromic microcytic anæmia. The irregularities within the gastric pouch are due to retained particles of food and not to gastric ulceration.

ANÆMIA

Seven patients were admitted to hospital for serious anæmias (*Table IV*). Though none of them volunteered a history of overt hæmorrhage there was no real difficulty in incriminating the gastro-intestinal tract. *Cases* 20 and 23 recalled passing dark stools coincidental with exacerbations of their illnesses, and in the other cases occult blood was found in the stools. Erect chest radiographs of 4 patients (*Cases* 20, 21, 22, and 23) revealed lower mediastinal shadows which were shown to be rolling hernias on barium study (*Fig.* 494). *Cases* 24, 25, and 26 had suffered from reflux dyspepsia, and *Case* 25 had experienced dysphagia for several years. These patients had sliding hernias complicated by œsophagitis and in one case by stricture. In none was a duodenal or gastric ulcer seen on radiological investigation.

Case 20 had been repeatedly advised to undergo surgery, and while in hospital he suffered a sudden massive hæmorrhage which persisted despite all measures. An emergency operation was done on the third day of hæmorrhage, the approach being from above the diaphragm, and a large incarcerated rolling hernia was found. The herniated stomach was thickened and hyperæmic, but no localized ulceration was felt. An Allison repair reinforced with nylon mesh was performed. Notwithstanding continuous gastric suction, the abdomen remained distended and bowel-sounds were only intermittently present. From the time of operation he complained of a dull ache in the left flank, and on the eighth day a laparotomy was done and gangrenous jejunum was found. The bowel was exteriorized and the abdomen drained, but the patient died soon afterwards. Unfortunately no necropsy was allowed, but it is unlikely that jejunal strangulation was the cause of the original anæmia. *Case* 22, a woman, was advised to have the hernia repaired, but resolutely refused. She has been observed for three years, and whereas for the first year she required frequent blood transfusions, she now no longer bleeds and has needed no transfusions for a year. This improvement has coincided with weight reduction. *Case* 23, also a woman, died

Table IV.—LIST OF CASES ADMITTED TO HOSPITAL BECAUSE OF UNEXPLAINED ANÆMIA

CASE	AGE (Years)	SEX	HÆMOGLOBIN	TYPE OF HERNIA	INCARCERATION	ŒSOPHAGITIS	OCCULT BLOOD IN STOOLS	TREATMENT	RESULT	FOLLOW-UP (Months)
20	49	M.	8·4	Rolling	+	0	+	I.H.	Death	—
21	62	M.	6·8	Rolling	+	0	+	D.H.	Cure	24
22	66	F.	8·2	Rolling	+	0	+	C.		30
23	59	F.	7·8	Rolling	+	0	+	Died before treatment		—
24	44	F.	10·4	Sliding	—	+	+	D.H.	Cure	10
25	43	M.	11·6	Sliding	—	+	+	D.H.	Cure	20
26	76	M.	10	Sliding	—	+	+	D.H.	Cure	4

I.H., Immediate herniorrhaphy. D.H., Delayed herniorrhaphy. C., Conservative.

suddenly from a massive pulmonary embolus while awaiting surgery. At necropsy an incarcerated rolling hernia was found. Incarceration was caused by the bulk of stomach wedged in the mediastinum and not by perigastric adhesions. So much stomach had undergone volvulus that the pylorus lay close to the hiatus. The anterior wall and lesser curve of the stomach had rolled into the peritoneal sac in front of the œsophagus, but the greater curve and fundus remained intraperitoneal. From without, no obvious abnormality of the stomach was visible. When the stomach was opened a line of demarcation between the normal pink mucosa of the greater curvature and pylorus and the dark reddened velvety mucosa within the herniated portion of stomach was seen. An oval superficial ulcer was present on the anterior surface close to the lesser curve about an inch from the cardia. This was the only possible source of hæmorrhage in the gastro-intestinal tract. The 3 patients with sliding hernias have had herniorrhaphies and are now well. *Cases* 24 and 25 were œsophagoscoped post-operatively when normal appearances were found.

There is little doubt that the 4 seriously anæmic patients with incarcerated rolling hernias were bleeding from the mucosa of the herniated stomach. This was proved at necropsy in one case and in another is strongly suggested by the cure which has followed herniorrhaphy. Pathologically, these cases are identical with the 10 cases of massive hæmorrhage complicating rolling hernias already described. The 3 patients with lesser degrees of anæmia were probably bleeding from the œsophagus as a result of digestive œsophagitis.

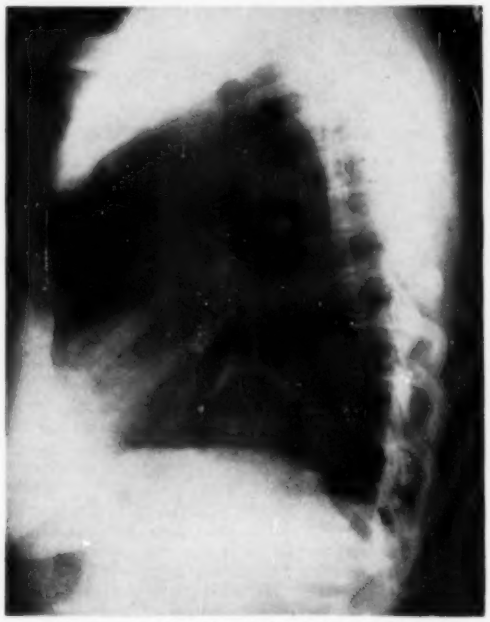

FIG. 495.—Erect lateral radiograph of the chest of a patient admitted with massive gastro-intestinal hæmorrhage. Note the fluid level within the mediastinum which denotes an incarcerated hiatus hernia.

FIG. 496.—Lateral chest radiograph of a patient with gastro-intestinal hæmorrhage. A solid rounded mediastinal tumour representative of an irreducible hiatus hernia is present.

MILD HÆMORRHAGE

Eleven patients have experienced minor hæmorrhages. None has had a hæmoglobin level of less than 13 g. per cent. Nine had small hæmatemeses (occult blood in stools positive in 3), and 2 had noticed dark fæces and occult blood was present in their stools. One of these had a large rolling hernia, the other an incarcerated sliding hernia with œsophagitis. All 9 patients with hæmatemeses had œsophagitis—5 with strictures—and 2 had associated duodenal ulcers. Except for 2 elderly women with strictures all have had operative reduction of their hernias (one œsophago-jejunostomy) and have had no further hæmorrhages, notwithstanding 2 recurrences of the hernia.

It is believed that the stomach was the source of hæmorrhage in the patients with the rolling and the incarcerated sliding hernia, that the œsophagus was the source in 7, and that duodenal ulcers may have been responsible in 2.

ANALYSIS OF CASES

Type of Hernia.—Severe hæmorrhage has predominantly complicated large rolling hernias where considerable amounts of stomach have passed into the mediastinum (*Table V*). The bulk of the hernia usually lies in front of the œsophagus within a peritoneal sac, but on occasions part of the stomach may protrude behind.

Mild hæmorrhages have usually complicated sliding hernias with reflux œsophagitis. The œsophagitis has invariably been severe and in 5 instances strictures have been present. Sliding hernias complicated by massive hæmorrhage have all had œsophagitis and duodenal ulcers; the latter have almost certainly caused the hæmorrhages. This was proved at one necropsy and, in 3 patients, ulcers were removed with the stomachs at emergency operations done by other surgeons. Hæmorrhages have not recurred, and the patients came under our care because of dyspeptic symptoms due to sliding hiatus hernias with peptic œsophagitis.

FIG. 497.—Erect barium radiograph of chest. An incarcerated rolling hiatus hernia is present. This case presented with severe anæmia.

Incarceration.—A hernia has been considered incarcerated on the following findings:—

1. A fluid level in the mediastinum on erect radiography (*Fig.* 495).
2. A persistent lower mediastinal shadow, either solid or air-containing, on straight radiographs (*Fig.* 496).
3. Demonstration of the hernia with barium swallowed in the erect position (*Fig.* 497).
4. Finding incarceration of the hernia at operation.

Ten of the 14 rolling hernias which bled seriously, and both the sliding ones, were incarcerated. In these, barium remained within the herniated stomachs for considerable periods of time, even with the patients ambulant between examinations. Five patients with rolling hernias had symptoms of high gastric obstruction. Attacks were intermittent, suggesting that the degree of incarceration varied as more or less bulk of stomach occupied the mediastinum (*Figs.* 498, 499). This is supported by the operative findings, where only in 4 cases were the herniated stomachs found to be fixed by perigastric adhesions. All the others were reducible by traction, and the amount of stomach above the diaphragm probably varied with posture, gastric distension, meteorism, abdominal compression, etc. Though perigastric adhesions were unusual, the hernias have usually remained within the mediastinum with the patient erect, and only in 4 cases did the stomachs reduce completely when the patient was ambulant. Even these were functionally incarcerated during recumbency, as shown by the delay in the passage of barium through the herniated pouch (*Fig.* 500). Many other patients have been similarly examined and it has been found that in the absence of incarceration, prolonged retention of barium within the supradiaphragmatic stomach pouch is unusual. Both sliding hernias which caused serious hæmorrhages were incarcerated. They were unusually large for sliding hernias, and passage of barium was delayed within them. Their distinction from rolling hernias as regards the production of hæmorrhage is academic, but the fact that the cardia formed the apex of the hernia, so that no œsophagogastric angle remained, allowed for free reflux of gastric enzymes into the œsophagus and the production of œsophagitis. At operation it was clear that the herniated gastric walls of both cases were abnormal and the probable sources of the hæmorrhage. These cases are important because they indicate that incarceration of the hernia is an essential factor in causing severe hæmorrhage. It is unusual for sliding hernias to be large, and consequently they are unlikely to impact because of the bulk of stomach lying above the diaphragm.

Table V.—ANALYSIS OF SEVERITY OF HÆMORRHAGE ACCORDING TO THE NATURE OF THE HERNIA AND THE CAUSE OF HÆMORRHAGE*

	Mild	*Severe*
Rolling hernias	1	14
Sliding hernias	12	2
Other associated causes	2	7

* Cases discussed under 'Anæmia' are included; hæmoglobins of more than 10 g. per cent are listed as mild and under 10 g. per cent as severe.

Cause of Hæmorrhage.—Duodenal or low gastric ulcers have caused 6 severe hæmorrhages and 2 minor ones. One patient died from hæmorrhage due to associated and probably unrelated œsophageal varices. The causes of hæmorrhage in these cases are clear and need no further amplification. Sixteen patients with severe hæmorrhage or anæmia and one with mild hæmorrhage were considered to have bled from the mucosa of the herniated stomach. In 4 cases ulcers were found within the trapped stomachs either at operation or necropsy. Five other gastric hernias were obviously abnormal; the walls were thickened and hyperæmic though no localized ulcers were palpable. The herniated stomachs of 6 patients were grossly normal when inspected at operation, but no alternative sources of hæmorrhage were found and repairs of the hernias have been curative. In the only necropsy case the stomach looked normal from without, but the thickened velvety mucosa of the herniated wall was clearly delimited at the level of the hiatal limbs, and close to the lesser curve a superficial erosion was present. Arguing from this evidence, the absence of ulcer induration or of any visible external abnormality does not detract from

the thesis that hæmorrhage has arisen from the herniated gastric mucosa. Twelve patients, with minor hæmorrhages, had sliding hiatus hernias complicated by œsophagitis, but not by other peptic ulceration or gastric incarceration. Where operation

FIG. 498.—Lateral barium radiograph showing relatively small rolling hernia during a period of remission of symptoms of gastric obstruction.

has succeeded in curing the œsophagitis, bleeding has not recurred. It is considered that in these cases the blood arose from the inflamed œsophagus.

DISCUSSION

Massive Hæmorrhage.—The findings of this review confirm the contention of Barrett (1950) that œsophageal erosion, due to gastro-œsophageal reflux, does not cause massive bleeding. Allison and Johnstone (1953) report 7 cases of "œsophagus lined by gastric mucous membrane"; 4 had gastric ulcers of the œsophagus, and 1 bled profusely. We have only 1 proved adult case of 'Barrett's ulcer', but this did not bleed. It would seem that massive hæmorrhage from an œsophageal ulcer associated with hiatus hernia only occurs in those rather rare cases where the lower œsophagus is lined by gastric mucosa, and the cause is then a true gastric ulcer. Blood which oozes from an area of reflux œsophagitis never more than stains vomitus, though its presence may be detectable by orthotolidine and amidopyrine tests of the stools.

The association of duodenal ulcer and hiatus hernia is well known. In this series only 6 massive hæmorrhages and 2 minor ones were due to low gastric or duodenal ulcers, but because gastro-intestinal hæmorrhages are first handled by physicians and general surgeons, it is inevitable that we should treat comparatively few cases where duodenal ulcers have been the cause. Of our total of 104 sliding hernias 25 (24 per cent) have been associated with duodenal or low gastric ulcers (18 did not bleed).

FIG. 499.—Lateral barium radiograph of same patient as in *Fig.* 498 taken under similar conditions of recumbency and shortly after a period of vomiting and upper abdominal colic. *Fig.* 498 was taken a week after *Fig.* 499. Note how the hernia has increased in size by further rolling of the stomach into the anterior hernial sac in front of the œsophagus. The appearances here are identical with those of *Fig.* 491, and are typical of all rolling hernias.

The incidence is sufficiently high to render it probable that massive hæmorrhage, in the presence of a sliding hernia, is due to a duodenal or low gastric ulcer and not to the hernia. On the other hand, only 2 of 34 rolling hernias were accompanied by duodenal ulcers (6 per cent); so that massive hæmorrhage, when associated with a rolling hernia, is usually a direct complication of the hernia itself.

Anæmia.—On rare occasions an anæmic patient will be exhaustively investigated before a causal hiatus hernia is found. More usually dyspepsia and overt hæmorrhage draw early attention to the stomach. Twenty-six patients (19 per cent) in the present series were anæmic at some stage, but in 19 it was melæna or hæmatemesis which precipitated admission to hospital. In 1946 Katzigras reviewed 955 published cases of hiatus hernia and found that anæmia was present in 80 (8·3 per cent); Schwartz (1950) described 20 cases and van Hoek and Musselman (1951) mention 36 patients with microcytic hypochromic anæmia where hiatus hernia was the cause. Recurrent and long-continued hæmorrhage or bleeding from the herniated gastric mucosa or from an inflamed lower œsophagus would readily account for the development of hypochromic anæmia, and upon the

amount and rate of the blood-loss would depend its severity. Certain authors have insisted that this concept does not explain every case. Chevallier and Danel (1944) were impressed by the refractory nature of the anæmia when treated with iron alone, and by the difficulty they experienced in demonstrating the presence of occult blood in the stools. They claimed better therapeutic results when treatment was supplemented with liver extracts and they

FIG. 500.—Radiograph of upper abdomen three hours after barium swallow, the patient having remained continuously in bed. Note retention of barium within the herniated pouch of stomach and clearance from the remainder of the stomach.

postulated that the anæmia is partly due to atrophy of the gastric mucosa and to gastric achylia. Coudinis (1949) supported this contention, because of his experience of a case of hiatus hernia complicated by microcytic anæmia for 24 years, which later converted to a profound hyperchromic macrocytic anæmia. Also, van Hoek and Musselman were only able to demonstrate occult blood in the stools of 21 (57 per cent) of 36 cases with progressive anæmia. All our cases have had hypochromic microcytic anæmias for which persistent blood-loss provides adequate explanation. Certainly it is the rolling hernia which is complicated by severe anæmia, the type also associated with severe overt hæmorrhage. One must expect anæmia, caused by continuous blood-loss, to be refractory to iron, repeated transfusions, vitamin B_{12} or liver extracts; for as long as the source of hæmorrhage remains, such treatment can only palliate. Surgical treatment of the hernia has proved curative in our patients who survived the operation.

Cause of Hæmorrhage in Rolling and Incarcerated Sliding Hernias.—There are 18 cases where neither the œsophagus nor the duodenum could have been the site of hæmorrhage. Ten rolling hernias caused massive hæmorrhage, 4 produced serious anæmia, and 1 mild bleeding; 2 incarcerated sliding hernias bled profusely, and 1 mildly. The evidence shows that in these cases even where no gross abnormality was found at operation, hæmorrhage was due to mucosal ulceration of the herniated stomach.

Gardner (1933) believed that blood oozes from the mucosa of incarcerated hernias because the venous drainage of the stomach is obstructed by compression of the edges of the diaphragm. Mendelsohn (1946) agrees with this explanation, as do Zarowitz and Graysel (1950). Harrington (1929) and Truesdale (1932) considered that hæmorrhage was due to mucosal ulceration of the supradiaphragmatic stomach and that this was caused by vascular disturbances. Belsey (1954), by histamine test-meal analysis, found a high, rapidly climbing acid curve associated with peptic ulceration within the intrathoracic stomach. He claimed that the post-reduction acid curve returns to normal or near-normal, and he believed that hyperacidity is responsible for the ulceration. Ten of our 18 bleeding rolling hernias were tested by the histamine technique, and 4 had high gastric acid levels which fell after operation. Our limited experience is therefore only partly in agreement with Belsey's. Aylwin (1953), in the course of an investigation into the causes of reflux œosphagitis complicating sliding hiatus hernias, analysed the fluid which regurgitated into the terminal œsophagus during sleep. Considerable volumes of secretion with a low content of protective mucus, a high pepsin concentration, and a *p*H usually well capable of activating the pepsin, were collected. He demonstrated, by introducing a dye into the stomach, that only juice secreted by the herniated pouch regurgitates into the œsophagus during sleep. This suggests some obstruction at hiatus level, which indeed was found in our patients with incarcerated hernias who were radiographed recumbent for several hours after swallowing barium. These observations suggest that with rolling hernias, secretions remain within the stomach throughout sleep and, if the hernia is incarcerated, stasis may even persist when the patient is ambulant. It is suggested that trapped enzymes digest the mucosa which has formed them, and that bleeding arises from acute or chronic gastric erosion. The theory of auto-digestion is supported by the fact that in all cases the pathological changes in the stomach wall were sharply limited at the level of the hiatus, suggesting that a functional block has existed here. The cause of retention within the herniated stomach is probably compression of the hiatal limbs aided by the effect of the differential pressures between chest and abdomen which are demarcated at this level (Marchand, 1958). It is unlikely that venous congestion is caused by constriction of the stomach by the hiatal edges, because the gastric veins communicate freely with the submucosal and extrinsic œsophageal veins and provide an abundant alternative route for the venous drainage. An additional factor probably operates when discrete ulcers are present, for although the whole mucosa of the gastric pouch is inflamed, the ulcer is always situated on or near the lesser curvature

close to the cardia. It is possible that the constricting edges of the hiatus traumatize the stomach here and so determine the site of ulceration.

Practical Considerations.—A hiatus hernia should be routinely considered, together with the more usual peptic ulcerations and œsophageal varices, whenever the problem of undiagnosed gastro-intestinal hæmorrhage presents. The hernia is either a large rolling one or, rarely, an incarcerated sliding one. The diagnosis cannot be excluded by the absence of a history of reflux dyspepsia. Rolling hernias may be silent, they may present with cardiac or pulmonary symptoms, or with a feeling of epigastric fullness related to meals and readily confused with duodenal ulcer dyspepsia, but reflux dyspepsia is rare. Many of these hernias are incarcerated or have undergone extreme volvulus so that gastric obstruction results. A history of vomiting with meals associated with epigastric colic is therefore common. Œsophagoscopy is of little help, for in the presence of active hæmorrhage interpretation is difficult. In any case, rolling hernias seldom produce œsophagitis and the cardia may not be displaced upwards under the conditions of the examination. The best pre-operative diagnostic measure is a plain, penetrating, postero-anterior and lateral chest radiograph to determine the presence of a mediastinal mass. This may contain a fluid level if the picture is taken erect, it may show an air cyst with thick walls, or it may be radiopaque (*Figs.* 495, 496). The absence of radiological evidence of ulceration within the hernia by no means excludes this as the source of hæmorrhage. If, at laparotomy done because of continuous hæmorrhage of unknown source, no obvious cause is found, the hiatus should be exposed before a 'blind' partial gastrectomy is considered. Hernias that bleed are usually large, but if traction is exerted on the stomach during the exploration the hernia may be unknowingly reduced and the stomach appear to be normally situated. This also applies when a para-cardial ulcer is found at laparotomy. In this event the surgeon may well be satisfied that he has found the site of hæmorrhage, as indeed he has, but the underlying cause of the ulcer may remain undetected if the hiatus is not explored. It is probable that many partial gastrectomies have been done in the past where the basic cause of the hæmorrhage has been a rolling hiatus hernia. This happened with at least one case presented here. The success of these blind gastrectomies in alleviating hæmorrhage is due to the reduction of the hernia during manipulation, particularly when attempts are made, as they frequently are, to deliver as much of the stomach as possible into the operative field. Subsequent recurrence of volvulus into the hernial sac, but not of the upward slide of the cardia, may be prevented by the excision of a large amount of stomach and the fixation afforded by the gastrojejunal or duodenal anastomosis. Finding a hernia does not necessarily mean that it is responsible for the hæmorrhage. Normally no peritoneal sac protrudes into the mediastinum, but by pushing firmly into the hiatus, it may be possible to evaginate the peritoneum slightly. Hernias that bleed are large, and before implicating the hernia a large defect admitting several fingers must be found. Should a characteristic defect be present without chronic ulceration within the stomach pouch, simple reduction of the hernia and repair of the hiatus should be undertaken. We have usually excised chronic ulcers either by wedge resection or partial gastrectomy, but herniorrhaphy alone may be equally curative.

If the pre-operative diagnosis of hiatus hernia has been made, I believe that the most satisfactory approach is through a thoraco-abdominal incision. The diaphragm is split in the line of the incision towards the hiatus, but stops short of the left hiatal muscle. The exposure allows complete assessment of the lower œsophagus, the stomach, and the duodenum, and permits dissection of the hiatus and accurate placing of reparative sutures. With large defects I support the hiatal limbs above by suturing a nylon mesh, perforated to accommodate the œsophagus, around the narrowed hiatus. All stabilizing œsophago-phrenic sutures are brought through the mesh and are tied over it, as are all interhiatal sutures. The mesh is not intended to bridge a gap, but to provide direct support to the hiatal muscles and also to fix the sutures, so that they are unlikely to tear out. A cuff of mesh is also sewn to the peri-œsophageal tissue and so helps anchor the terminal œsophagus. This technique has been used on 16 large hernias and there have been no recurrences. However, the follow-up is short and the cases too few to make any definite claims for the efficacy of this method of repair.

SUMMARY

Thirty-seven cases of hæmorrhage out of a total series of 138 cases of hiatus hernia (27 per cent) are described. Seven of these patients were admitted to hospital with unexplained anæmia, 11 had experienced mild hæmorrhage at some stage in the disease, and 19 had bled massively. Most patients with massive hæmorrhages or severe anæmia had large rolling hernias. All massive hæmorrhages and severe anæmias not caused by duodenal ulcers were due to ulceration of the mucosa within the herniated stomachs. Œsophagitis has only caused mild bleeding.

The possible factors responsible for gastric ulceration within the hernia are discussed. Incarceration of the hernia is usual and it is believed that autodigestion by trapped gastric enzymes is the main cause. Traumatization by the constricting edges of the hiatus may be responsible for discrete ulceration.

A plea is made for considering the possibility of an incarcerated rolling hernia in any case of massive unexplained gastro-intestinal hæmorrhage. The diagnosis can usually be established by means of penetrating postero-anterior and lateral radiographs of the chest. A 'blind' partial gastrectomy should never be performed for hæmorrhage unless the hiatus has been adequately explored.

A technique for reinforcing the hiatal limbs after a standard Allison repair of the hiatus is briefly described.

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MESOCOLIC FIBROSARCOMA

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INTRODUCTION

IN a review of the regional characteristics of fibrosarcomata, Stout (1948) has expressed the opinion that the behaviour of these tumours is to some extent influenced by their site of origin. Further, that fibrosarcoma occurred uncommonly in the mesentery, and that metastasis must be an exceedingly uncommon event.

Because of the paucity of completed case records relating to metastasizing fibrous tumours arising in unusual sites, a case of mesocolic fibrosarcoma which came to autopsy is described.

Since the mesenteries of the small and large bowel share a common embryological origin for the median dorsal mesentery (Hamilton, 1956), the behaviour of certain tumours that arise from them might prove comparable.

Therefore, from the literature on solid mesenteric tumours some case-histories have been studied with a view to establishing the general pathology and behaviour of these fibromatous tumours.

CASE REPORT

Mrs. V. P. 18061. An obese mother of three children, she enjoyed good health until December, 1951. Then, a few weeks short of her forty-sixth birthday, she was peeling potatoes and suddenly felt a severe pain down the left leg which prevented her from standing or sitting. For this she was referred to an orthopædic surgeon. A radiograph of the spine showed scoliosis and secondary arthritic change, and a diagnosis of sciatica and lumbago due to prolapsed intervertebral disk was made and physiotherapy prescribed. Because attacks of pain continued a plaster jacket was made in February, 1953, and later a surgical corset was worn.

In April, 1955, the patient again visited her doctor complaining of attacks of pain in the left loin radiating to the left iliac fossa. These pains were worse at night. A lump was then felt in the left iliac fossa, and the patient having recently experienced the climacteric, she was referred to hospital for a further opinion. The lump was not freely mobile and was not associated with any disturbance of bowel habit. Since the nature of the lump was not apparent, laparotomy was advised.

AT OPERATION.—Laparotomy was performed (Mr. H. E. Lockhart-Mummery) in May, 1955, and what was considered to be a colloid carcinoma of the proximal end of the sigmoid colon was found. It was adherent to the left psoas muscle, and a biopsy of this site was taken. Left hemicolectomy was performed and the transverse colon was anastomosed to the remnant of the sigmoid. The patient was well when discharged a month later.

PATHOLOGY.—

Macroscopic Surgical.—A sharply-defined, lobulated, soft-tissue tumour projected from an adipose pelvic mesocolon. It measured 11 × 9 × 10 cm. and was adherent to a segment of adjacent colon; 32 cm. of colon had been excised, and the whole specimen weighed 575 g. (*Fig.* 501). The mucosa of the colon was normal in appearance, and freely mobile upon its muscle coat. There was no demonstrable pathological relationship between it and the tumour. The muscle coat was, however, tethered to the tumour at the point of contact.

On section, the tumour was either solid, firm, opaque white, and whorled, or softer grey and glistening. These areas were haphazardly mixed. A small satellite nodule was found nearby.

Microscopic Surgical.—The tumour had a thin, incomplete collagenous capsule and was separated from the intact muscularis of the colon by a thin zone of fibrous connective tissue. The firmer areas were moderately cellular and consisted of interlacing bundles of spindle cells with long polar fibrillary processes which stained well with Heidenhain's iron hæmatoxylin. The nuclei were usually blunt-ended, hyperchromatic, or coarsely stippled with chromatin and karyosomes. Two or more mitotic figures were present in high-power fields and atypical X and Y forms were seen. Pleomorphism was evident in some areas, particularly at the growing edge of the tumour, where binucleated tumour giant cells were sometimes seen (*Fig.* 502).

The tumour produced copious fibre which stained with aniline blue and acid fuchsin, and which was distributed as wisps or trabeculæ. With silver impregnation a fine tangle of fibrils surrounded individual cells.

The softer areas were mucoid and much less cellular. The stroma stained positively with Southgate's mucicarmine, strongly with the periodic-acid–Schiff technique; and metachromasia was evinced with thionin. A few lymphocytes and plasma cells were found in these mucoid areas.

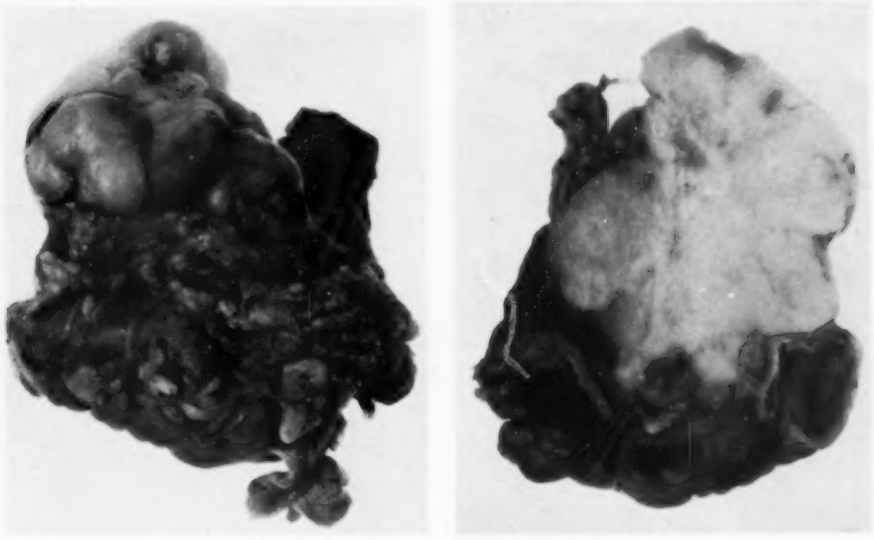
Blood-vessels within the tumour were scanty and thin walled. Frozen sections did not reveal lipoid vacuoles within the tumour cells.

A few small peripheral nerve-fibres averaging 150 μ in diameter were also present at the edge of the tumour. These showed no histological abnormality. Further tissue removed at the time of operation consisted of a piece of tumour adherent to psoas fascia. This showed microscopical infiltration of adipose tissue and of the sheath of a small nerve-fibre. A small para-aortic lymph-node showed chronic inflammatory changes only.

A diagnosis of mesocolic fibrosarcoma was reported.

PROGRESS.—Within five months of the operation the patient was back in hospital complaining of increasing breathlessness. This had recurred and persisted after a sudden fit of coughing a week previously, for which she had been confined to bed.

She was found to have collapse of the right lung and bronchoscopy revealed a pedunculated tumour arising from the right main bronchus. Its removal completely relieved her symptoms and a palliative dose of radiotherapy to the carina was given. The tumour was sectioned and a histological diagnosis of fibrosarcoma was made.



A B

FIG. 501.—A, External view of primary mesenteric tumour with related segment of pelvic colon. B, Cut surface view of primary mesenteric tumour with related segment of pelvic colon. ($\times \frac{1}{10}$.)

A B C

FIG. 502.—Photomicrograph of primary mesenteric tumour. A, Low-power view to show tumour growing up to the muscularis of the colon. (H. and E. × 15.) B, and C, High-power views to show cellular pleomorphism and mitotic activity in fibroblasts. Heidenhain's iron hæmatoxylin and orange G. (× 600.)

An examination of this section was later made available (*Fig.* 503) through the kindness of Dr. G. M. Hamilton of Harefield Hospital. Most of the tissue showed hæmorrhagic necrosis with fresh thrombus in thin-walled blood-vessels. Where viable tumour was present this showed widely-spaced spindle cells in a fine fibrillary matrix giving a syncytial appearance. Pleomorphism was very

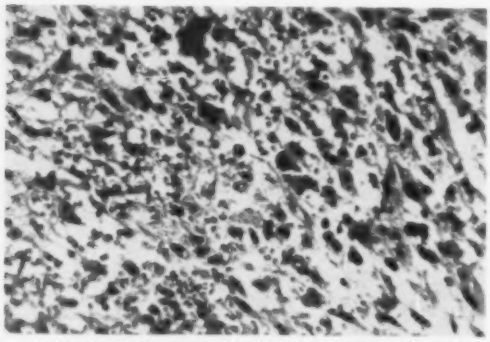


FIG. 503.—Photomicrograph of the tumour arising from the right main bronchus. Tumour has undergone marked anaplasia. (Section supplied by courtesy of Dr. G. M. Hamilton.) H. and E. (×300.)

marked and giant-cell forms frequent. Mitotic figures averaged less than 1 per high-power microscopic field.

The patient's general health then began to fail, with loss of energy, appetite, and weight. By March, 1956, severe abdominal pain had become constant and the abdomen began to swell. Her general condition rapidly worsened. She died in hospital during April, aged 50 years, eleven months after hemicolectomy was performed.

AUTOPSY.—April 24, on the third day after death.

The body was that of an obese middle-aged woman with the scar of a left paramedian incision upon a distended abdomen. There was pitting œdema of both lower limbs up to the thighs. Post-radiation pigmentation of the neck was present; elsewhere the skin was normal.

A smooth lobulated tumour arising out of the left iliac fossa filled the abdomen and displaced viscera. It was up to 30 cm. in diameter, weighed 6570 g., and was firmly attached on the left side to the iliopsoas muscles, pelvic and parietal peritoneum of the anterior abdominal wall. A few coils of small intestine adhered loosely to the tumour, but the colon was stretched, though not ulcerated, on its left side.

The tumour was solid, fleshy, and friable, glistening white, yellow, or red. The colic anastomosis had healed and the rectum contained fæces. The only tumour in the gastro-intestinal canal was one small subserosal nodule in the ileum. Pressure on pelvic veins had induced thrombosis. Thrombus filled the lower limb veins, but did not extend into the inferior vena cava. There was no ascites.

Metastatic deposits were found as follows (*Fig.* 504):—

Liver.—A few scattered spherical nodules in both lobes of the liver; some were just visible beneath Glisson's capsule. The largest tumour was in the left lobe and was 3 cm. in diameter.

Pancreas.—Scattered nodules were embedded in the body of the pancreas and were not visible from external surface.

Thyroid.—A 1-cm. diameter nodule was in the upper pole of the right lobe of the gland.

Kidneys.—Scattered nodules occupied the cortex of both kidneys, up to 0·5 cm. in diameter.

Lungs.—Present in all lobes were a few scattered nodules 0·5 cm. in diameter; those beneath the surface caused pleural puckering. The air-passages were now clear and the lungs filled the chest, although they were consolidated and congested.

Skeleton.—The tumour infiltrated the marrow of the vertebral bodies of T.11, L.3 and 5, without causing their collapse. Sternal bone-marrow was tumour-free.

Lymph-nodes were nowhere enlarged and were apparently tumour-free. The heart was small, the spleen soft and pink. No significant pathology was noted in the genitalia or nervous system.

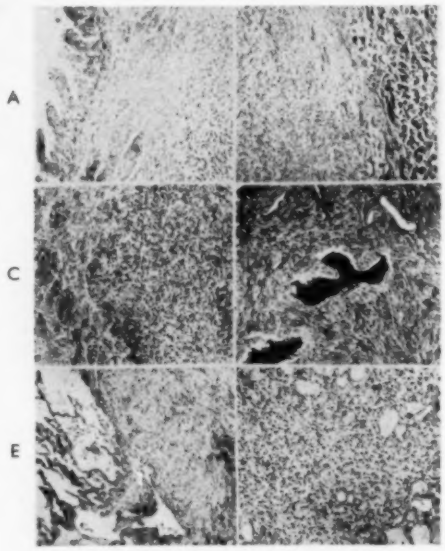


FIG. 504.—Photomicrographs of secondary deposits seen at post-mortem. A, Serosa of small intestine. B, Liver. C, Kidney. D, Spine. E, Lung. F, Thyroid. H. and E. (×50.)

Microscopically the tumour was more cellular and less well differentiated than the original tumour; less fibre and no mucin were produced.

DISCUSSION

In a paper entitled "Fibromatous Tumours of the Mesentery", Greer (1911) reported the enucleation of a fibromyxoma from the mesentery of the small intestine which weighed over 3 lb. The patient was a man aged 54 years. The author referred to the state of chaotic obscurity that existed on this subject before the nineteenth century, and to the difficulties of histological diagnosis subsequently. Nevertheless, he collected some 33 cases from the literature, dating back to 1824, and tabulated them. Amongst these were 3 cases of mesocolic tumours in adults of both sexes. They were called "lobulated fibroma", "hæmorrhagic fibroid myxomatous masses", and "calcified fibroma". In general these tumours were considered to be slow growing and unlikely to recur after removal. The largest tumour in the series that was successfuly removed weighed over 40 lb.

Similarly, Harris and Hertzog (1897), giving brief details, tabulated 57 cases of solid mesenteric tumour dating from 1730. Only one case of fibrosarcoma is mentioned, and it is attributed to Zirndorf.

In 1920 a fibrosarcoma as large as a fœtal head was excised from the mesosigmoid with a foot of colon (Ransohoff and Friedlander, 1921). The

tumour was discovered during the examination of the abdomen of a 73-year-old man. It had caused a deformity of the sigmoid loop which was demonstrated radiologically after a barium enema. Recurrence was noted thirteen months later.

Fisher's (1929) patient was a 37-year-old woman who for two years complained of iliosacral backache and of pain referred down the thighs to the knees. This was relieved by surgical belts designed to minimize spinal movement, and finally cured by the enucleation of an encapsulated fibromyoma of the pelvic mesocolon, 9 cm. in diameter. The author quotes Szenes (1918), who in an analysis of 60 cases of mesenteric tumour found that fibroma accounted for 29 per cent of this total and fibrosarcoma for 13 per cent.

Wakeley (1931) regarded tumours of the mesentery as sufficiently rare to be regarded as pathological curiosities. He recorded a case of a man aged 54 years who had a large, hard, fibroma in the small intestine mesentery. The tumour was removed and the patient died on the fifth post-operative day. Autopsy was performed, but there was no further reference to the primary condition.

Gordon-Taylor (1931) reported a case of fibrosarcoma of the jejunal mesentery in a woman of 38 years, but gave no follow-up details.

Eight malignant tumours of the mesentery were extracted from the files of the Mayo Clinic by Rankin and Major (1932). Their *Case* 17 was that of a man aged 54 years who had an inoperable fibrosarcoma of the small intestine mesentery; *Case* 18 was a cystic fibrosarcoma that perforated the small intestine of a man aged 44 years. In a 53-year-old man with an ileal tumour, metastases were reported in the liver some six years after surgery (*Case* 20). Unfortunately this tumour was loosely called a spindle-cell sarcoma. This case illustrates both the necessity for recording an accurate histological diagnosis, and for observing the subsequent progress of the patient for a number of years.

Again, Walters and Priestly (1934) recorded a sarcoma of the mesosigmoid in a man aged 34 years, but neither the nature of the tumour nor the fate of the patient was recorded.

Weaver (1933) described a "massive cellular fibroma of the transverse mesocolon" in a Negro girl aged 10 years. This tumour weighed 1820 g. and was attached to 20 cm. of normal colon. This patient is known to have remained well for about one year after operation. Summers (1932) and Bruton (1936) report further pædiatric cases.

Amongst 163 cases of fibrosarcoma of soft parts Warren and Sommer (1936) mention in *Table* 10 two cases that arose in the mesentery. *Case* 3 was that of a man aged 40 years who was treated by surgery and irradiation. He died of recurrence without metastases a little over a year later. *Case* 6 went untreated. The patient was a man aged 38 years, and died of his disease in less than six months without metastases. The tumour was a neurogenic fibrosarcoma.

In the same year a comprehensive review of the literature, including 186 cases of solid tumours of the mesentery, was presented by Hart (1936). Amongst these were brief notes on 1 fibroma, 2 fibrosarcomata, and 1 spindle-cell sarcoma, all of the mesocolon. He discussed the symptomatology, and advocated a watch for recurrences for up to ten years. His own case was in a man of 46 years who had a 5½-lb. tumour in the mesentery of the small intestine. This tumour had undergone central liquefaction and was called a fibrosarcoma, but the diagnosis of the sections was not agreed amongst other pathologists to whom they were shown.

Professor R. A. Willis examined the tumour reported by Nicholas (1947), and although the cut surface suggested a fibrosarcoma, microscopy showed a tumour of low cellularity and much collagen. This tumour arose in the small intestine mesentery of a man aged 34 years. Subsequent progress is not fully recorded.

Stout (1948) reported 6 cases of fibrosarcoma of the mesentery from his Columbia University series. All 6 cases were well-differentiated tumours displaying infiltrating growth, and resected with adjacent small bowel. Of the 4 cases that were followed up, there was one instance of local recurrence removed after two years, and none of metastasis. Stout concluded that "metastasis must be an exceedingly uncommon event".

Ackerman (1954) tabulates 15 cases of primary malignant tumour of the mesentery seen between 1905 and 1951 at Columbia University. The 6 cases of fibrosarcoma recorded are in fact attributed to Stout (1948). Delannoy, Martinot, and Soots (1958) report 4 cases of sarcoma of the mesentery and mesocolon, and state that there are another 200 cases in the literature. Their *Case* 3 was that of a woman aged 54 years; she had a large fibromyxosarcoma of the transverse mesocolon removed. She died three months after excision, and during this time two hard, plum-sized tumours appeared in the abdominal wall in the region of the scar. Their other cases included recurrent fibromyxosarcoma of the mesocolon from a primary omental tumour, polymorphic fibrosarcoma of the mesentery, and a case of a fibroliposarcoma of the transverse mesocolon. In their experience the prognosis was bad.

Mesenteric neurofibromatosis has been reported by Barton and Inglis (1931), in a 40-year-old man who presented with intestinal bleeding. The lesion was in the ileal mesentery and presumably benign. There were associated cutaneous tumours.

Stewart and Copeland (1931) advanced the view that a large proportion of sarcomata arising in soft parts are of neurogenic origin and that recurrence is the rule after excision. They described many cases where the tumour was solitary; the symptoms complained of were those due to a mass which was sometimes painful.

Stewart and Copeland, Inglis (1950 a, b, c), and Willis (1958) review the stigmata associated with von Recklinghausen's disease, and if any of these are present, the likelihood of a fibromatous tumour being of neurogenic origin is increased. In the absence of these stigmata either in the patient or relatives, the histological appearances of such a tumour may not be sufficiently characteristic to enable a diagnosis of neurogenic fibrosarcoma to be established (Winston Evans, 1956; Willis, 1958).

Thus, from the literature that has been reviewed here, it would seem to emerge that fibrous tumours of the mesenteries are rare; that they arise more commonly in the mesentery than in the mesocolon, but

behave in a similar manner; that they grow slowly, but sometimes to massive proportions in members of both sexes, including children. Many cures are reported following surgical excision of the tumour with or without resection of a segment of intestine, but it should be noted that most of the cases were not followed for a period of years.

In one of Stout's cases there was a recurrence within two years of operation, and Warren and Sommer's *Case* 3 died with a recurrence within a year. Ransohoff and Friedlander reported a recurrence thirteen months after operation. In Delannoy and others' *Case* 3, two tumours appeared in the abdominal wall within three months of the operation. (These tumours do not seem to have been examined pathologically; and there are no post-mortem reports in this fatal case, nor in that of Warren and Sommer's *Case* 3.) Rankin and Major's case of spindle-cell sarcoma developed metastases after six years. Inoperable cases were reported by Warren and Sommer and Rankin and Major.

However, the histological features of the primary tumour in the case now presented were those of a rapidly growing, infiltrating, cellular neoplasm with evidence of anaplasia in the form of pleomorphism, frequent and abnormal mitotic figures, and tumour giant cells. Anaplasia was even more marked in the secondary deposits examined histologically after bronchial biopsy and autopsy. These microscopical observations were reflected in a clinical course of only one year from the time that the mass was first discovered. (In Warren and Sommer's experience these histological features are associated with a liability to recurrence and to metastasis and with an average survival time of two years from the onset of symptoms.)

Thus within five months of hemicolectomy, a polypoid mass was found occluding a main bronchus and causing massive collapse of the lung. This phenomenon has been described by Willis (1952). The combination of bronchoscopic removal and radiotherapy relieved this complication, and at autopsy no trace of that tumour could be found.

The place of radiotherapy in fibrosarcoma is controversial.

Thus, in Stout's 1948 series of fibrosarcomata, radiotherapy was used successfully in 3 out of 37 cases of recurrence after surgery. It is of interest that one of these was a case of primary fibrosarcoma of the trachea in a woman of 28 years, who afterwards remained well for six years.

In Warren and Sommer's series its use was described as disappointing. Radiotherapy was employed in some of the cases in Delannoy and others' series, but the authors were not dogmatic about the indications for this. No doubt much will depend upon the size and location of any particular tumour.

Massive local recurrence within the abdomen was an outstanding feature in this case. The tumour had grown outside the peritoneum and had remained fairly well circumscribed while obstructing the venous return from the lower limbs. Whereas the metastases found at autopsy were widespread in viscera and bone, these had not dominated the clinical picture. Although lymph-node spread in fibrosarcoma has been reported by Warren and Sommer, this is unusual and did not occur in the present case. This behaviour trait is of some importance to the surgeon when resecting these tumours.

Finally, it may be said that the absence of any alteration of bowel habit is an important clinical point in the diagnosis of mesenteric tumours that have not actually caused intestinal obstruction or ulceration.

SUMMARY

The surgical, pathological, and post-mortem findings in a case of fibrosarcoma of the mesocolon are described and discussed with reference to other reported cases. Notable features were the absence of bowel symptoms; early massive recurrence after resection; widespread metastases in viscera and bone, but none in lymph-nodes.

Acknowledgements.—I am indebted to Mr. R. S. Murley and Mr. H. E. Lockhart-Mummery, under whose care this patient was originally treated, for encouragement and permission to publish this case; and to other colleagues at St. Albans City Hospital who were also consulted. I thank Dr. C. S. Pike and Dr. Cuthbert Dukes for reading and criticizing this text. The histological sections and photographs were prepared under the direction of Mr. T. R. West.

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SPONTANEOUS DISAPPEARANCE OF LUNG METASTASES IN A CASE OF KIDNEY CARCINOMA (HYPERNEPHROMA)

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THE following case seems worthy of record, as only rarely are malignant metastases observed to regress spontaneously.

CASE REPORT

J. C., a bus driver, aged 55 years, presented in November, 1955, complaining of swelling in the left side of the scrotum for a year, undue fatigue for three months, and a cough for years, recently productive of yellow sputum. On examination the following abnormalities were found: A left varicocele, a grossly enlarged palpable left kidney, a blood-pressure of 200/120, and Hb 18·3 g. (124 per cent). The E.S.R. was 1 mm./hr. (Wintrobe), and microscopy of the urine revealed no abnormality. Radiography of the renal area revealed calcification in the lower pole of the enlarged left kidney. The diagnosis was confirmed by intravenous pyelography, which showed the right kidney to be excreting normally. The ascending pyelogram was also confirmatory (*Fig.* 505). Radiographs of the thorax at this time showed an opacity in the right lower zone suggestive of a secondary deposit.

FIG. 505.—Ascending pyelogram showing calcification in the lower pole of the enlarged left kidney.

Eighteen days after his first attendance a left nephrectomy was carried out (M. F. N.) with uneventful recovery. The nephrectomy specimen, which included the suprarenal, showed the lower pole of the kidney replaced by a growth 13 × 11 × 8½ cm. which had grown into the lumen of the renal vein. There was also an entirely discrete nodule of growth in the upper part of the kidney. Microscopy showed the growth to be a clear-cell carcinoma mostly of tubular structure, but papillary in some areas (*Fig.* 506); the discrete nodule showed similar microscopic appearance. Microscopy also revealed a second discrete deposit in the suprarenal gland of similar cell type.

A few weeks after nephrectomy, consideration was given to removing the secondary deposit in the right lung. This was postponed, as there was doubt whether there were not multiple shadows in the lung, and as two presumably blood-borne secondaries had been demonstrated in the removed kidney and suprarenal.

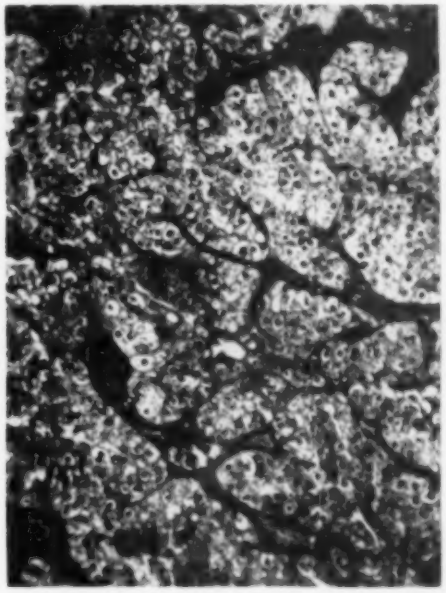
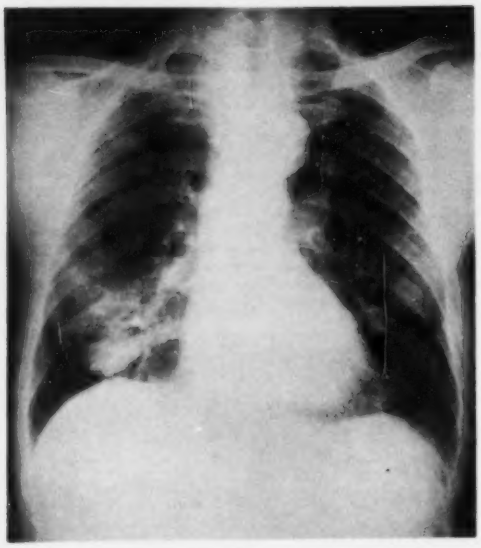


FIG. 506.—Clear-cell carcinoma, mostly of tubular structure, papillary in some areas. H. and E. (× 190.)

Two and a half months after the nephrectomy there was no clinical evidence of further secondary deposits and there was only one really definite shadow in the lung fields (*Fig.* 507). An exploratory right thoracotomy was therefore done (A. H. M. S.). Apart from a mass about 4 cm. in diameter in the lower lobe, at least a dozen smaller nodules were felt scattered throughout the lung. No material was taken and thus no microscopical proof of the nature of the deposits was obtained. Recovery from this exploratory operation was uneventful. A chest radiograph taken two weeks after this operation was the last to show the large shadow in the lower lobe. Three months later a postero-anterior film of the chest failed to reveal a definite lung shadow, and in many subsequent postero-anterior and lateral films no trace of the shadow was present. Eighteen months after the last film showing the shadow, tomographic cuts showing the whole thickness of the chest showed no trace of any deposits in the lung fields (*Fig.* 508).

The patient remained in reasonable health and without clinical or radiological evidence of metastasis for eighteen



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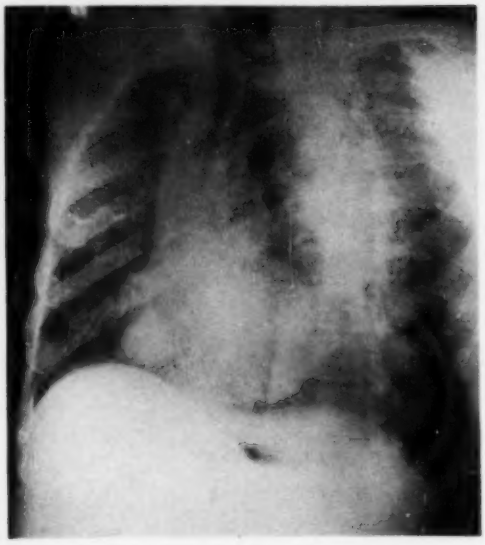

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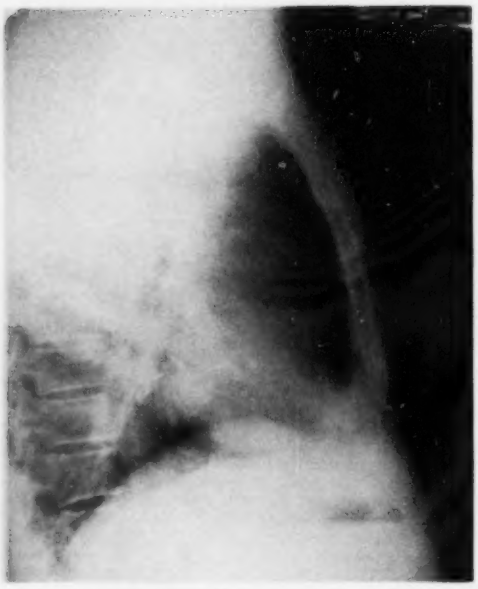

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FIG. 507.—A–C, Radiographs of chest showing secondary deposit in right-lower lobe.

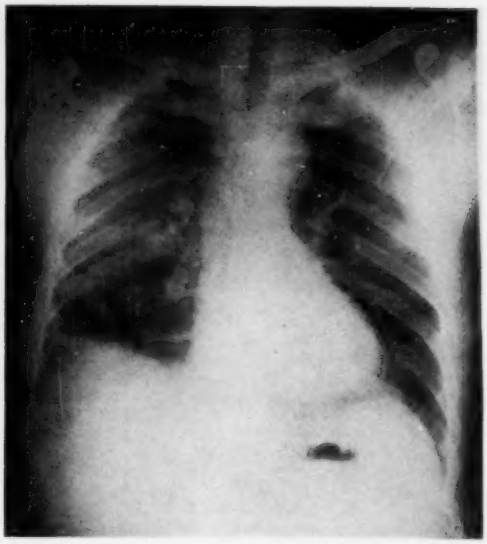

FIG. 508.—Radiograph of chest taken in June, 1957; no trace of any deposits in the lung fields.

months after the nephrectomy, when he complained of pain in the right buttock, soon extending down the leg. As radiographs of the lumbar vertebræ and sacrum at this stage appeared normal, he was treated, on the assumption that he might have a disk lesion, with rest and later a corset. Two months later a radiograph of the sacrum revealed a secondary deposit which steadily extended in spite of radiotherapy. The patient was given a maximal skin dose of 2000 r (factors: 250 kV., 50 cm. F.S.D., 1·9 mm. Cu H.V.L.). This treatment gave little relief of pain and he was kept in hospital, the growth in the sacrum being observed to extend gradually, involving eventually lumbar vertebræ and forming a pelvic mass. There was also evidence of nerve-root involvement. He required increasing sedation and steadily deteriorated to death two and a half years after the nephrectomy.

At no time had he been treated with hormones or any other drug likely to affect the course of the malignant disease. During the last six months of his life his Hb level varied from 14·9 (114 per cent) to 14·5 (98 per cent).

Autopsy showed extensive infiltration of the retroperitoneal tissues extending to and involving the seminal vesicles. The growth had a vascular polypoid appearance. The right kidney showed compensatory hypertrophy. The lumbar vertebræ and sacrum were extensively invaded by growth involving the cauda equina. Microscopy showed this to be the same clear-celled type of carcinoma.

The lungs showed no evidence of secondary deposits or other abnormality except mild emphysema on extensive sectioning.

The brain was not examined, but other organs showed no significant abnormality.

SUMMARY OF CASE-HISTORY.—A man aged 55 years presented with a left renal tumour and shadow suggesting a secondary in the right lung. Nephrectomy revealed a carcinoma with apparently blood-borne metastases in the removed kidney and its suprarenal. Two and a half months later right exploratory thoracotomy revealed multiple masses in the lung which were not removed. These are believed to have been secondary deposits.

For a year the patient remained symptom-free. During this time the deposits in the lung which had been visible on radiography disappeared. Full tomography failed to reveal them, and eventually at post-mortem examination no trace of secondary deposits in the lungs could be found.

A year after nephrectomy a secondary deposit appeared in the sacrum and gradually extended in spite of radiotherapy. Death occurred two and a half years after nephrectomy. Post-mortem examination revealed secondary deposits in the lumbar vertebræ and sacrum involving the pelvis and cauda equina.

COMMENT

Regression or disappearance of carcinoma has occasionally been reported. Everson and Cole (1956) analysed a series of 600 cases, of which in their opinion 47 had been absolutely proved. Of these only 2 were renal carcinoma. Recently Hallahan (1959) has reported a remarkably similar case, in which pulmonary metastases from a renal carcinoma regressed spontaneously following nephrectomy.

In the case here reported the histological proof is not complete, but in our opinion it is otherwise firmly established.

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THE USE AND RISKS OF THE ROUX-EN-Y ANASTOMOSIS IN THE RELIEF OF POST-GASTRECTOMY SYMPTOMS

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POST-CIBAL regurgitations of bile, or intermittent vomiting of larger quantities, in association with other symptoms of the so-called afferent loop syndrome, constitute one of the main disadvantages of gastric resection with a gastrojejunal anastomosis.

There are variable reports of the incidence of these symptoms and the degree of severity is not always defined. In Visick's series (Pulvertaft, 1952) of 512 men with primary resection for ulcer, 18·4 per cent (7·4 per cent severe) complained of bilious regurgitation; this compares with 15·2 per cent, severity not defined, reported by Goligher, Moir, and Wrigley (1956), and 23·3 per cent (9·6 per cent severe) of a composite series of 1406 patients analysed by Capper and Welbourn (1955).

Minor degrees of bilious regurgitation cause only slight inconvenience which can often be modified by dietary measures; when severe, however, the symptoms are likely to be incapacitating, rendering life miserable for the patient, and cannot be controlled by dietary or other medical measures.

The use and value of the various surgical procedures which have been suggested for the relief of the syndrome were discussed by Capper and Welbourn. This report included a series of 15 cases of simple entero-anastomosis between afferent and efferent limbs carried out in this clinic, the initial results of which were encouraging, but there was a high relapse rate within eighteen months from operation. The same report also referred to two series of conversions to Y anastomosis (15 in York and 13 by Schofield in Swindon).

Tanner (1951) referred to the use of Roux-en-Y conversion after total gastrectomy, the patient being relieved of bilious regurgitation, but drew attention to risks of stomal ulceration if it was used after a partial resection—a reputation that it had earned in the past, mainly because of its then use in conjunction with gastro-enterostomy or low gastric resections. Information about such risk when used as a secondary procedure with subtotal resections is, however, limited, though Schofield and Anderson (1953) reported 3, and Wells and Johnson (1956) 21 conversions with excellent symptomatic results, and no recurrent ulcers; the periods of follow-up were, however, short.

This paper concerns the use and risks of Y anastomosis when used as a secondary procedure; it relates to a consecutive series of 54 conversions (45 men and 9 women) performed between April, 1951, and September, 1955, the minimum follow-up being two and a half years. Unfortunately, one patient was lost, due to coronary occlusion, the day following operation. These patients have been selected from a total follow-up of some 1200 people.

In the majority of patients (38 men and 8 women) the indications for revision were for one or more of the following symptoms: frequent post-prandial bilious regurgitation, intermittent and copious bile vomiting, severe nausea or heartburn. We have not

regarded 'dumping' symptoms, i.e., post-prandial palpitations, perspiration, headache, dizziness, or limb weakness, as indications for surgery, although a number of patients did experience some degree of one or more of such symptoms, in addition to the above.

The remaining 8 patients (7 men and 1 woman) form a miscellaneous group with more varied indications (*see below*).

group. The risk is therefore small, but may be rather greater than appears from these figures, as a woman with a two-thirds resection for duodenal ulcer, but not yet seen for two and a half years since revision, has a probable, but so far unconfirmed, jejunal ulcer. Vagotomy was performed at the time of conversion in some cases, but not routinely. It is, however, acknowledged that it would be advisable as an additional precaution.

Table I.—Details of Original Resection

Previous Ulcer	Men				Women			
	Extent of Resection at Gastrectomy				*Extent of Resection at Gastrectomy*			
	Two-thirds to three-quarters	Sub-total	Unknown	Total No.	Two-thirds to three-quarters	Sub-total	Unknown	Total No.
Duodenal ulcer	10	23	3	36	2	1	1	4
Stomal ulcer	2	0	0	2	0	1	0	1
Gastric and duodenal ulcer	0	0	1	1	0	0	0	0
Gastric ulcer	1	4	0	5	1	1	2	4
Total	13	27	4	44	3	3	3	9

Table II.—Length of Follow-up since Y Anastomosis in Patients with Previous Duodenal or Stomal Ulceration

Time (years)	Men				Women			
	Extent of Resection at Gastrectomy				*Extent of Resection at Gastrectomy*			
	Two-thirds to three-quarters	Sub-total	Unknown	Total No.	Two-thirds to three-quarters	Sub-total	Unknown	Total No.
2½	11*	23	4	38	2	2	1	5
3	11*	20	4	35	1	2	1	4
4	7	12	1	20	0	2	1	3
5	5	3	0	8	0	1	0	1
6	0	2	0	2	0	1	0	1
7	0	1	0	1	0	0	0	0
Total	11	23	4	38	2	2	1	5

Note: 1 patient with two-thirds resection (duodenal ulcer) died before 2½-year period.

* 1 patient developed stomal ulceration.

RESULTS

The value of this procedure has been judged by the following:—

1. The risk of recurrent ulceration.
2. General clinical improvement.
3. Relief of specific symptoms.

1. Risk of Further Stomal or Jejunal Ulceration.—This is likely to vary with: (1) The original type of ulcer. (2) The extent of resection.

The details of original resection are given in *Table I*. Thirty-nine men and 5 women had the primary resection for either a duodenal or stomal ulcer.

The length of follow-up of patients with previous duodenal or stomal ulceration is shown in *Table II*.

To date, only one stomal ulcer has occurred, in a man with a previous two-thirds resection for duodenal ulcer; it developed within a year of revision. No further ulceration has been seen in the gastric ulcer

2. General Clinical Improvement.—This has been judged by alterations in 'mean grading' and weight-gain.

All patients are seen at periods of six months and classified according to the York system (Visick, 1948; Pulvertaft, 1952). The average grade before and after revision can, therefore, be compared. A mean grade of 1–2 implies an excellent result, with only very minor symptoms that are easily controlled; 2·1–3 a moderate result, patients experiencing symptoms which are not always controlled by minor modifications in dietary habits, but are improved as compared with their pre-operative condition; 3·1–5 indicates a bad result.

The results are summarized in *Table III*. Before conversion to Y anastomosis, 26 men and 5 women were regarded as bad results (3·1–5); of these, 13 are now 'excellent' and 17 'moderate'; 1 remains a failure. This is due to stomal ulceration, which has

required two further resections, but now with an almost total gastrectomy the patient is symptom-free.

Ten men and 3 women were classified initially as 'moderate' results; 6 are now 'excellent', 6 unchanged, and 1 has deteriorated to a 'failure'.

Table III.—Comparison of Pre- and Post-conversion Mean Grades

	No.	Post-en-Y Mean Grade		
		1–2	2·1–3	3·1–5
1. Patients graded as bad results before revision (3·1–5)				
Men	26	12	13	1*
Women	5	1	4	0
Total	31	13	17	1
2. Patients graded as moderate results before revision (2·1–3)				
Men	10	6	4	0
Women	3	0	2	1
Total	13	6	6	1

* Further stomal ulceration.

One man has been unable to attend the clinic and, although he reports that he is greatly improved, is excluded from this assessment.

The over-all picture is that of a general clinical improvement, which in many instances is dramatic. The main reason that a number of patients are still classified as only 'moderate' results is the persistence of epigastric fullness and vasomotor symptoms.

Weight-gain.—The majority of patients gain weight after conversion, but the initial gain is not always fully maintained; for this reason, the weights at the most recent assessment have been compared with those immediately prior to conversion. Such comparative weights are available in 30 men and 7 women (*Table IV*). A gain of half a stone or more is regarded as significant; by this standard 21 have gained, and of these 9 men and 2 women have gained more than 1 stone.

Table IV.—Effect on Weight

Comparison of Present Weight with that before Revision

	Men	*Women*
No change	11	5
Gain of half a stone or more	10	0
Gain of 1 stone or more	9	2
Total	30	7

3. Effect on Specific Symptoms.—There is a marked variation in the severity of each symptom, e.g., a patient may complain of frequent and copious bile vomits, with relatively little nausea, or alternatively of severe nausea with only infrequent and small bile refluxes. The symptoms are, therefore, classified as severe or moderate.

The results of Y conversion are summarized in *Table V*.

Bile Reflux or Vomiting.—Twenty-three patients (20 men and 3 women) complained of severe bile reflux or bile vomiting; 17 are completely cured (of these, 1 later developed food vomiting and another occasional regurgitations of bile after extra indulgence); 3 still experience occasional symptoms and 3 have not benefited. Seventeen patients (12 men and 5 women) experienced moderate symptoms; 15 are completely cured (one man vomits food very occasionally). One remains unchanged, and 1 has developed food vomiting.

Table V.—Results of Y Conversion

	Men				Women			
		After Revision				*After Revision*		
	No.	None	Moderate	Severe	No.	None	Moderate	Severe
1. Severe symptoms								
Bile vomiting or reflux	20	16*	1	3	3	1	2	0
Nausea	18	11	4	3	3	1	2	0
Heartburn	5	5	0	0	2	1	0	1
Epigastric fullness	25	4	13	8	3	0	2	1
Vasomotor symptoms	7	3	1	3	3	0	0	3
2. Moderate symptoms								
Bile vomiting or reflux	12	12†	0	0	5	3	1	1‡
Nausea	7	5	2	0	5	3	1	1
Heartburn	0	0	0	0	1	0	1	0
Epigastric fullness	9	4	4	1	4	1	3	0
Vasomotor symptoms	12	4	8	0	0	0	0	0

* Of these, 1 later developed food vomiting, and another vomits bile on very rare occasions.
† Vomits food occasionally.
‡ Food vomiting.

Nausea.—Twenty-one patients (18 men and 3 women) complained of severe nausea; 12 are completely cured, 6 improved, and 3 unchanged. Of 12 with less severe nausea, 8 are completely cured, 3 unchanged, and 1 now reports severe nausea.

Heartburn.—Seven patients (5 men and 2 women) complained of severe heartburn; 6 are completely cured and 1 unchanged. One woman with occasional heartburn is unimproved.

Epigastric Fullness.—Twenty-eight patients (25 men and 3 women) complained of severe symptoms; 4 are completely relieved, 15 are improved, and 9 unaffected. Of 13 patients (9 men and 4 women) with less severe symptoms, 5 are cured, 7 unchanged, and 1 man reports increased symptoms.

Vasomotor Symptoms.—Ten patients (7 men and 3 women) reported frequent and severe symptoms; 3 are cured, 1 improved, and 6 unchanged. Twelve men reported slight symptoms; 4 are cured and 8 reported no improvement.

Miscellaneous Group.—The revision was used in an attempt to improve nutrition in 2 patients with advanced tuberculosis whose condition was deteriorating; it was not successful, both patients dying within one and a half years of revision.

Three patients (2 men and 1 woman) had complained of a variety of symptoms, regarded as psychoneurotic manifestations, since resection ten years before. The symptoms included bile vomiting, but the operation was used in an attempt to improve their general condition; in no instance was it successful.

In 2 patients the predominant feature was epigastric fullness, a discomfort amounting to pain which, although vomiting was not pronounced, was thought to be of afferent loop type. One patient was greatly improved and gained over a stone in weight, the other derived no benefit.

The remaining patient was seen by a psychiatrist, who considered that the basis of his symptoms was organic. Revision was performed, but no success was obtained.

DISCUSSION

The results suggest that the Y anastomosis is of real value in the relief of bile reflux and afferent loop symptoms; further, that the risk of secondary ulceration is small, even when vagotomy has not been performed routinely. The miscellaneous group illustrates that it is not a panacea for all post-gastrectomy troubles, and that good results are only obtained when indications are clear and well defined.

Symptoms most benefited are bile reflux or vomiting, nausea, and heartburn; the least influenced are epigastric fullness and the vasomotor complex.

It is thought that some of the less satisfactory results in the series may be due to an error in our technique, as during the early years the afferent loop was inserted only some 3–4 in. below the stomach remnant; it is now considered that this distance should be at least 10 in.

Epigastric fullness or discomfort can be due to distension of either the stomach, the afferent or the efferent loop; the Y anastomosis is, therefore, only likely to give relief when afferent loop distension is predominant; this probably explains why only 9 out of 41 patients obtained relief from this symptom.

Wells and Johnson (1956) reported that 3 patients were cured of 'dumping' symptoms. In the present series, only 7 out of 22 reported any relief. We have tested several patients with hypertonic glucose, and have found that both the symptoms and electrocardiograph changes are as easily reproduced after revision as before. According to present hypotheses of the aetiology of this complex, it is unlikely that the Y conversion would have any effect. Nevertheless, some patients do report improvement and we suggest that they were not suffering from the true post-gastrectomy vasomotor syndrome, but that the symptoms were the result of the severe upper abdominal discomfort and nausea, which even with an intact stomach may well be accompanied by pallor and sweating, etc.

If the secondary Y anastomosis is effective, it may be argued, as suggested by Schofield and Anderson (1953), that it should be incorporated as a primary procedure. This, however, does imply a high resection (or a complete vagotomy) as otherwise the risk of stomal or jejunal ulceration is known to be considerable. It was, in fact, used in this clinic for a short period, but was abandoned as 3 of 35 duodenal ulcer patients developed stomal ulceration within eighteen months of operation. We suggest, therefore, that it is better retained as a secondary procedure for the patients who require it.

It is generally accepted that the majority of stomal ulcers develop within two years of gastrectomy; for this reason and because some bile vomiters recover spontaneously, we suggest that, normally, conversion to Y anastomosis should not be considered under two to three years from the time of the primary resection.

SUMMARY AND CONCLUSIONS

The results of 54 (45 men and 9 women) secondary conversions of Polya gastrectomies to Y anastomosis for afferent loop symptoms, with follow-up for two and a half years, are reported.

In 39 men and 5 women the primary gastrectomy was performed for either duodenal or stomal ulceration; to date, only 1 man has developed stomal ulceration since conversion. No stomal ulcers have been seen in the 5 men and 4 women with primary gastric ulcers.

The value of the procedure has been assessed on:—

1. Clinical Grading.—Before conversion 31 patients were classified as 'bad' results; after conversion, 13 are 'excellent', 17 improved, and 1, owing to stomal ulceration, remains unimproved; 13 patients were classified as 'moderate' results; 6 are now 'excellent', 6 'moderate', and 1 'bad'.

2. Weight-gain.—Twenty-one of 37 'conversions' have gained half a stone or more.

3. Relief of Specific Symptoms.—The symptoms most improved are bilious reflux or vomiting, nausea, and heartburn; those least affected are epigastric fullness and vasomotor complex.

These results suggest that Y anastomosis is an effective procedure in the treatment of the afferent loop syndrome. It should not normally be considered for at least two years after the primary gastrectomy.

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LIPOSARCOMA OF THE GREATER OMENTUM

By W. A. T. ROBB

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MALIGNANT tumours of fatty tissue are rare. The following case of liposarcoma of the greater omentum is reported because of the unusual manner of clinical presentation.

CASE REPORT

A male aged 34 years was admitted as a surgical emergency to the Royal Infirmary, Edinburgh, on April 28, 1958, with a history of constant dull aching epigastric pain of forty-eight hours' duration. Two hours before admission the pain suddenly became much more severe, spread downwards towards the right iliac fossa, and then over the whole abdomen. There was no history of trauma, and prior to the onset of this illness he had always been healthy; in particular, he had never suffered from dyspepsia nor had he ever experienced previous attacks of abdominal pain.

ON EXAMINATION.—The patient was a fit-looking man whose general condition was good. Pulse-rate was 76 per minute, temperature 98·4° F., and blood-pressure 120/78 mm. mercury. There was restricted movement of the abdominal wall on respiration, generalized abdominal tenderness, and board-like muscular rigidity, most marked in the epigastrium and right side of the abdomen. On auscultation it was noted that bowel-sounds were absent.

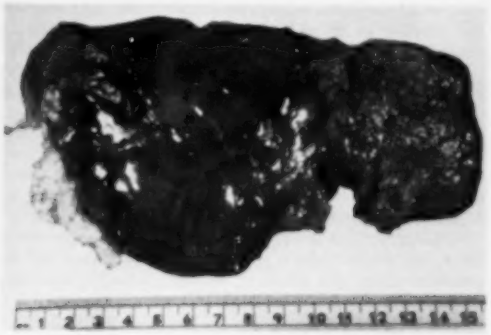

FIG. 509.—Anterior aspect of tumour showing site of rupture.

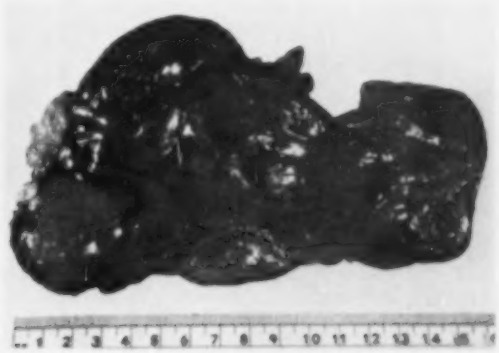

FIG. 510.—Posterior aspect of tumour.

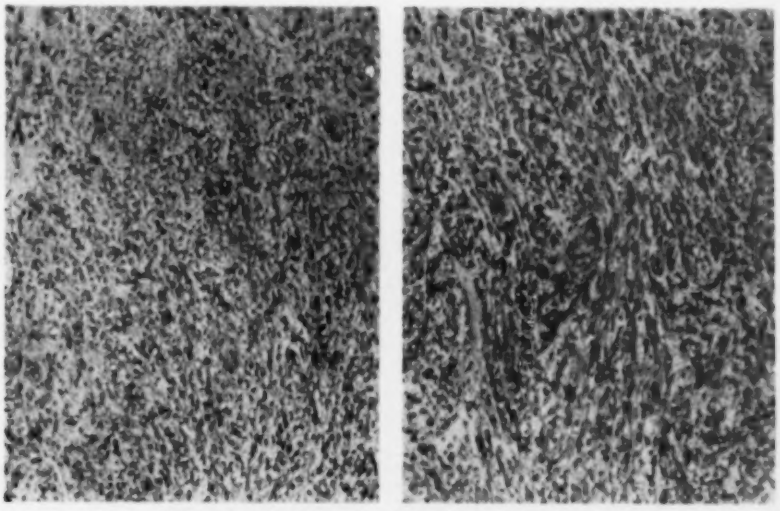

FIG. 511.—Histology of tumour showing a highly cellular neoplasm with considerable pleomorphism. (× 90.)

A clinical diagnosis of generalized peritonitis due to perforation of a peptic ulcer was made and preparation begun for operation.

AT OPERATION.—Under general anæsthesia (Dr. M. C. MacQueen) with pentothal, scoline, cyclopropane, and oxygen, the abdomen was opened through an upper right

paramedian incision. There was bright red blood in the peritoneal cavity, and immediately the peritoneum was opened the blood welled up into the wound at such a rate that at first it was difficult to determine its origin. It was considered possible that bleeding was occurring from a peptic ulcer which had also perforated, but the anterior wall of the stomach and duodenum showed no abnormality. The wound was therefore extended and further exploration showed that massive hæmorrhage was occurring from the rupture of a very vascular tumour situated in the greater omentum. The upper edge of the tumour, which was well defined, was 5 cm. from the greater curvature of the stomach and was quite separate from the transverse colon, so that its removal by excision of the whole of the greater omentum presented no difficulty.

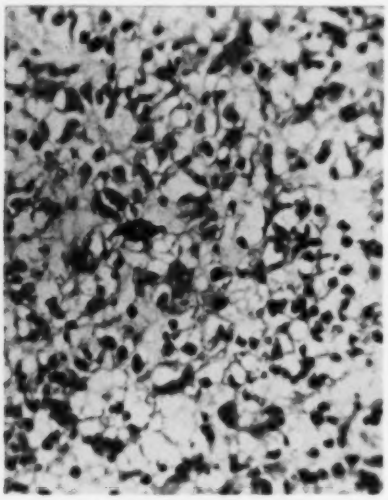

FIG. 512.—Histology of tumour showing presence of fat globules in the tumour cells. (× 270.)

A considerable quantity of free blood was sucked from the peritoneal cavity and it was estimated that the patient had lost in all over 2 pints of blood. This volume was replaced by transfusion.

During the laparotomy the lower pole of the spleen was lacerated and because of continued ooze of blood from this site splenectomy was performed. The liver was normal, there were no enlarged glands, and no evidence of tumour elsewhere in the abdomen. The wound was closed in layers without drainage.

POST-OPERATIVE PROGRESS.—The patient developed a post-operative paralytic ileus which responded to gastric suction and intravenous infusion. A post-operative chest radiograph showed no abnormality. The patient was discharged from hospital on the seventeenth day after operation. At the time of his last attendance as an out-patient, six months after operation, he was symptom-free and physical examination was negative.

MORBID ANATOMY.—The specimen (*Figs.* 509, 510) measured 16 cm. in its transverse axis, 8 cm. from above downwards, and 6 cm. in depth. It appeared dark red in colour, very vascular, lobulated, with cystic areas filled with blood. The margin was well defined.

Histological Report (Dr. A. W. Williams).—"The tissue is that of a malignant mesenchymal tumour, which has a pleomorphic picture and is partially undifferentiated but presents fields with the histological features of a liposarcoma. It is a highly cellular neoplasm containing a rich supply of blood-vessels, and the cells, which are arranged irregularly and loosely, or else in small perivascular, compact masses, or in ill-defined bundles, are of variable size and shape; some are spheroidal or spindle-shaped, whereas many are polyhedral, and many contain fat droplets which stain with Sudan III." (*Figs.* 511, 512.)

DISCUSSION

The sudden onset of symptoms, the absence of any previous history, and the abdominal findings which simulated those of a general peritonitis from a perforated peptic ulcer, are the outstanding clinical characteristics of the case reported. The symptomatology, the findings on physical examination, and the hæmoperitoneum found at laparotomy can be attributed to spontaneous rupture of the highly vascular tumour. Although the initial hæmorrhage had been insufficient to produce the features of shock due to blood-loss, when the peritoneum was opened the release of intra-abdominal pressure resulted in dramatic hæmorrhage.

Ransom and Samson (1934), who reviewed 75 primary tumours of the greater omentum, found that abdominal pain, a palpable tumour, ascites, weakness and loss of weight, abdominal distension, bowel irregularity, and anæmia were the common symptoms, in decreasing order of frequency. The ascitic fluid was usually blood-stained, which attests to the vascularity of most of the primary tumours of the omentum.

Primary tumours of the greater omentum are rare. In 1936 Menne and Birge considered that of 83 reported cases not all were proved to be instances of primary tumour, nor were they all correctly classified as to cell type.

The histological diagnosis and the determination of the true incidence of liposarcoma are difficult because of the wide variation in histological structure that may be encountered in these tumours. As Knowles and Huggill (1954) indicate, the relationship between differentiation and origin of malignant mesenchymal tumours is not always as close as in many other classes of malignant tumours where the structural differentiation exhibited by the tumour cells is a reliable guide to the tissue of origin. Proliferating connective tissue cells can give rise to a wide range of metaplastic processes with the frequent occurrence of mixed mesenchymal tumours. Willis (1953) warns against calling a tumour a liposarcoma merely because it contains fat, and emphasizes that the presence of fat-laden cells may be simply an indication of fatty degeneration of some of the cells of a tumour. For a diagnosis of liposarcoma there must be enough differentiation of the tumour cells to indicate their origin.

In the present case the diagnosis of liposarcoma is based on the characteristic lobulated structure of the tumour, the presence of fat globules in the tumour cells, the considerable pleomorphism, and mitotic activity with the observation of intermediate forms between undifferentiated sarcoma cells and those containing fat globules.

Liposarcomas may occur wherever fat is present, but unlike benign lipomata which occur most frequently in subcutaneous tissue, the malignant tumours are situated most commonly in the deeper tissues. The limbs and retroperitoneal tissues are the commonest sites (Knowles and Huggill, 1954; Stout, 1953). Of 105 cases of liposarcoma collected

over a period of twenty-five years at the Memorial Cancer Center, New York, Pack and Pierson (1954) found that the lower extremity was involved in 62·7 per cent, the upper limb in 24 per cent, and the retroperitoneum and trunk in 13·3 per cent.

SUMMARY

A case of hæmoperitoneum due to rupture of a liposarcoma of the greater omentum is reported. The sudden onset of severe abdominal pain and the tender rigid abdomen were the striking clinical features. The findings at operation and the nature and histology of the tumour are discussed.

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RENAL TUBERCULOSIS

LONG-TERM REVIEW OF CASES FOLLOWED-UP FROM TWENTY TO THIRTY-FIVE YEARS

BY R. I. HARRIS, W. K. KERR, AND H. S. COULTHARD

TORONTO HOSPITAL FOR TUBERCULOSIS

IN our community a long-standing interest in tuberculous bacilluria associated with bone and joint tuberculosis has provided an exceptional opportunity to study the course of renal tuberculosis over long periods of years. Harris (1929, 1930) reported the occurrence of tuberculous bacilluria in adult and juvenile patients suffering from tuberculosis of bones and joints, and advanced reasons for believing that the tubercle bacilli in the urine came from renal lesions of tuberculosis. The adults in this group were all soldiers of the first world war under treatment in Christie Street Hospital. Since that time they have been under the supervision of the Department of Veterans' Affairs. Their medical records are complete and available. The fate of all of them is known, and the survivors are accessible for study. The children in this group were patients in the Hospital for Sick Children, Toronto. Their records are available and it has been possible to locate some of the survivors and to learn something about those who died.

In 1930 an orthopædic department and a genito-urinary service were established in the Toronto Hospital for Tuberculosis, Weston, and there the study of renal tuberculosis was continued by the methods reported by Harris in 1929, later supplemented by improved techniques (e.g., intravenous pyelography) as these were introduced into genito-urinary surgery. At first the routine search for evidence of genito-urinary tuberculosis was limited to patients suffering from bone and joint tuberculosis (extrapulmonary lesions). Later, the investigation was expanded to include pulmonary cases which had no extrapulmonary lesions. This routine study has been continued to the present day and remains in charge of the persons who initiated it. Consequently, all the cases of renal tuberculosis cared for at Weston are known personally to the authors. The patients' records are available and their fate is known. The survivors are still seen in the follow-up clinic.

We have, therefore, two groups of patients available for a long-term study of the course of renal tuberculosis: (1) the adults and children reported in 1929, and (2) the cases studied at Weston since 1930.

This study was prompted by a desire to ascertain the fate of the cases reported by Harris in 1929. The availability of the cases from Weston added numbers to the review so that they were included also.

The original paper in 1929 reported the finding of 16 cases of tuberculous bacilluria among 43 adult patients with bone and joint tuberculosis, and 9 cases of tuberculous bacilluria among 67 cases of bone and joint tuberculosis in children. The incidence of 37·2 per cent of tuberculous bacilluria in adults and 13·4 per cent in children seemed surprisingly high. The explanation lay in the painstaking and repetitive search for tubercle bacilli in the urines of these patients. Specimens were collected every two months throughout their stay in hospital, without regard to the existence or absence of symptoms of renal tuberculosis, and were inoculated into guinea-pigs. Frequently, this routine of repeated search for tubercle bacilli revealed their presence in every specimen of urine from patients who were free of symptoms suggestive of genito-urinary tuberculosis.

Table I.—SOURCE OF 82 CASES OF TUBERCULOUS BACILLURIA

	Cases
Christie Street Veterans' Hospital, 1922–9	
Orthopædic Service	16
Hospital for Sick Children, 1922–9	
Orthopædic Service	9
Toronto Hospital for Tuberculosis 1930–5	
Orthopædic Service	36
Urological Service	21
Total	82

Though the study of the Weston cases has been continuous since 1930, only those cases which came under observation and treatment between 1930 and 1935 have been included in this survey. Thus they compose a group which has been under observation for not less than twenty years and up to twenty-five years. Combined with the cases reported in 1929 a

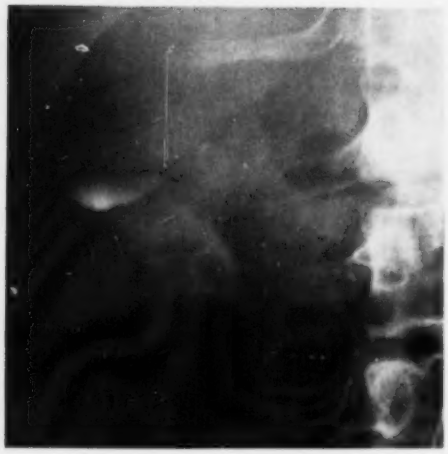
total of 82 cases is available for study (*Table I*). These patients received no antibiotics.

DISTRIBUTION OF RENAL TUBERCULOSIS

These 82 cases are not a specially selected group, but comprise all cases of renal tuberculosis encountered prior to January 1936 at the hospitals mentioned, with the exception of 14 cases of renal tuberculosis, treated at Weston by unilateral nephrectomy. In this latter group tuberculosis of the remaining kidney was not proved, though it was strongly suspected in some.

A

B



C

FIG. 513.—Mr. Y. A, Pyelogram, January 1936; B, I.V.P., June 1955, 30-minute film; C, Same I.V.P., June 1955, upright film. In 1933, aged 15 years, he developed bone and joint tuberculosis and tuberculous bacilluria which was also present in 1934 (bladder and right ureter), 1935, 1936, 1937, 1942, and 1955. In 1956 urine became tubercle-free on drug treatment. Gross progression of disease is evident.

Therefore they were unsuitable for inclusion with the cases of proven tuberculosis of the kidney. Seven of these patients are living to-day. One patient with tuberculous epididymitis underwent left orchidectomy and right nephrectomy in 1934. Thereafter no tubercle bacilli were recovered from the urine, so that this case was not included in the study. But in 1956 the patient died of uræmia and was found at post-mortem examination to have tuberculosis of the left kidney.

The following case was excluded, but the story is of some interest:—

Mrs. L. P. was cystoscoped (in Cray Valley Hospital, England, by Sir William Bennett) in 1912 for dysuria, and was found to have ulceration of the bladder. This was treated in hospital for five months and she was told that it was due to tuberculosis. Tuberculin injections were used. On Jan. 19, 1940, she was admitted to Weston for treatment of pain and burning on urination, a complaint which had been present for thirty years. Cystoscopy elsewhere in 1939 had shown bilateral renal tuberculosis. This was verified in 1940 and acid-fast bacilli were still present in the urine in 1942. In 1944 and again in 1956 urine showed albumin 1 plus or 2 plus with gross pus, but no tubercle bacilli, and intravenous pyelograms showed gross hydronephrosis and destruction of the right kidney and no function in the upper half of the left kidney. The patient has been working and supporting herself since 1941 and now is 63 years old, and still subject to intermittent attacks of cystitis, though urea nitrogen is only 18·0 mg. per cent. She probably has had renal tuberculosis for forty-four years.

Table II.—EXTENT OF RENAL TUBERCULOSIS IN 20-YEAR SURVIVORS

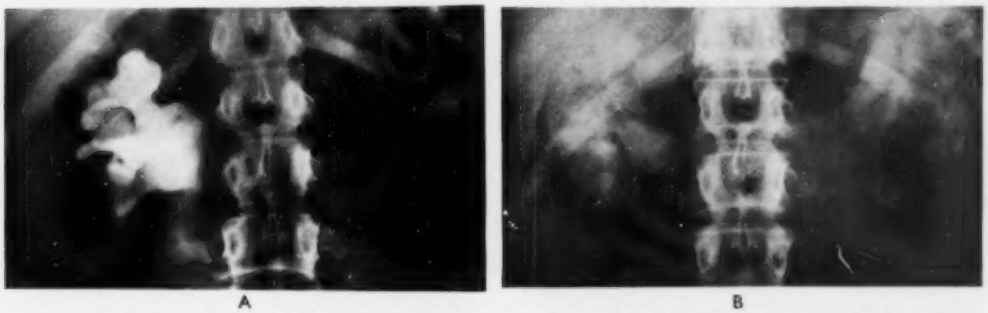
	Cases
Unilateral renal tuberculosis—no nephrectomy	5
Bilateral renal tuberculosis—no nephrectomy	6
Bilateral renal tuberculosis—nephrectomy done	8
Uncertain (not lateralized)—no nephrectomy	12
Total	31

EXTENT OF RENAL TUBERCULOSIS IN 5–18-YEAR SURVIVORS

Unilateral renal tuberculosis—no nephrectomy	2
Bilateral renal tuberculosis—no nephrectomy	5
Bilateral renal tuberculosis—nephrectomy done	8
Uncertain (not lateralized)—no nephrectomy	4
Total	19

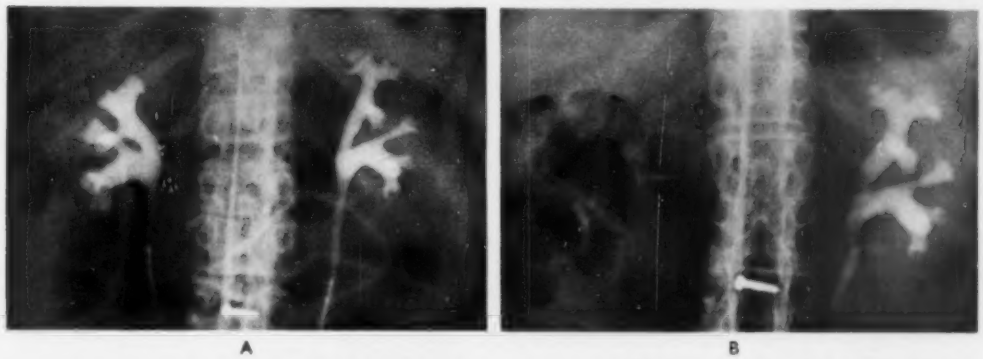
There were 31 cases which survived for twenty years or more after the diagnosis of renal tuberculosis was made. The distribution of their renal disease is shown in *Table II*.

Almost one-third of the patients are alive and well to-day, an average of 25·4 years after the diagnosis of renal tuberculosis was made. If to this is added the 6·0 per cent who survived for 20 years before dying, then 37·8 per cent of the 82 cases lived for 20 years, while 39·0 per cent died in less than 5 years. The remaining 23·2 per cent died between 5 and 20 years after diagnosis, with an average survival time of 10·3



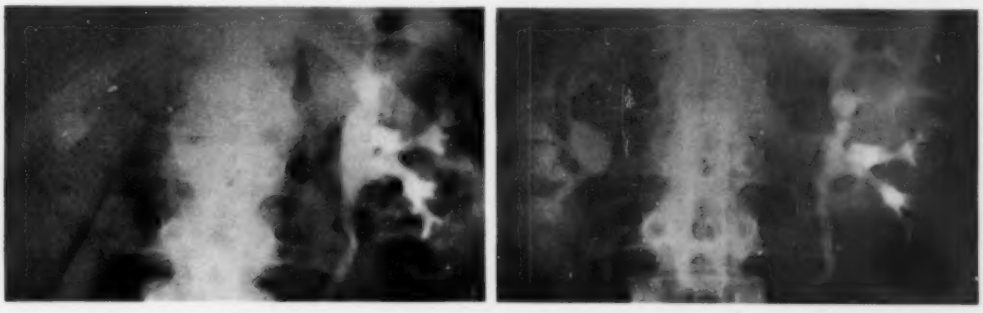
A B

FIG. 514.—Mrs. F. A, Pyelogram, April 1935; B, I.V.P., August 1956. In 1916 she had bone and joint tuberculosis at age of 11 years; in 1930 left nephrectomy for tuberculosis, and tubercle from the remaining right kidney in 1931, 1934, and 1935. She is known to have had pus and blood in her urine in 1948, 1949, and 1956, but no cultures were done. Radiographs now show atrophy of the hydronephrotic kidney of 21 years ago.



A B

FIG. 515.—Mrs. H. A, Pyelogram, June 1936; B, I.V.P., May 1955. In 1930, aged 21 years, she developed Potts' disease, and in 1936 left renal tuberculosis, with positive urine in 1937, 1938, 1939, and 1940. In 1941 the right kidney also became positive for tubercle and continued so in 1942, 1946, and 1953, when right nephrectomy was done. In 1955 urine was tubercle-free after drug treatment. Radiographs show small lesion of left superior calix which appears static over 19 years.



A B

FIG. 516.—Mr. M. A, Pyelogram, September 1936; B, I.V.P., August 1955. In 1920 this 22-year-old man had bone and joint tuberculosis, and tuberculous bacilluria was demonstrated then and in 1931, 1936, 1937, 1942, 1949, 1954, and 1955. Since drug treatment in 1955 the urine has been tubercle-free. The marked hydronephrosis in 1936 probably exaggerated the true extent of disease which is radiographically barely evident 19 years later in 1955.

years. This was without the aid of chemotherapy. (*Table III.*)

Table III.—Final or Latest Status of 82 Cases of Tuberculous Bacilluria

	Cases	*Per cent*	*Average Survival in Years*
Alive and well in 1956–7 (20–35 yr.)	24	31·7	25·4
Alive and well in 1951–2 (25–26 yr.)	2		
Died after surviving for 20 years	5	6·1	21·2
Died between 5 and 20 yr. after diagnosis	19	23·2	10·3
Died within 5 yr. of diagnosis	31	39·0	1·9
Lost within 1 yr. of diagnosis	1		
Totals	82	100·0	

The degree of tuberculous renal disease in the group surviving for 20 years or more was determined radiographically in 15 out of the 26 living cases. Of the 15 pyelograms, 11 showed definite but small lesions, while 4 were normal.

CAUSES OF DEATH IN 82 CASES OF TUBERCULOUS BACILLURIA

There are 26 patients living to-day, and one patient was lost one year after diagnosis, when he went to live in a home for the aged and was not followed up. The remaining 55 patients have died, a mortality-rate of 66·3 per cent including those followed for 20 to 35 years. Very few of these patients were free from active tuberculosis at the time of death, though 2 are definitely known to have been so, one dying from cerebellar tumour, and one from gas asphyxiation. The only other deaths listed as non-tuberculous were acute coronary occlusion in 3, myocardial decompensation in 1, and 'terminal congestion' in 1. None of these occurred within the first 5 years, during which time *non-renal* tuberculosis caused two-thirds of the deaths, and renal tuberculosis one-third. After that first 5-year period almost half the deaths were due to renal tuberculosis (11 out of 24), one-quarter (6) to non-renal tuberculosis, and one-quarter (7) to non-tuberculous causes. (*Table IV.*)

Table IV.—Causes of Death in 55 Cases*

Survival Time	Total Number	Renal Tb.	Non-renal Tb.	Non-tuberculous
20 yr. and over	5	2	0	3
5–19 yr.	19	9	6	4
0–5 yr.	31	11	20	0
Totals	55	22	26	7
Percentage of 82	66·3	26·5	31·3	8·4

* Among the non-renal lesions causing death were pulmonary tuberculosis 12 cases, meningitis 8 cases, amyloid disease 3 cases, miliary tuberculosis 2 cases, and Addison's disease 1 case.

FINDINGS IN 26 CASES LIVING IN 1951–7

It has been possible to have examinations done on 21 of 26 survivors. These findings are tabulated in *Table V.* Urine examinations were made on 17, while cultures were done on 16, one of which was contaminated and had to be discarded. Two patients whose urine cultures were positive in 1955 became negative in 1956 and 1957 after receiving chemotherapy. Intravenous pyelograms were made in 1956 and 1957 on 11 cases, and in 1949 on one other which was said by the patient to be normal.

Table V.—Present Findings in 26 Patients still Living

Signs or Symptoms	*Present*	*Absent*	*Total*
Frequency or burning	9	12	21
Pus in urine	5	8	17
Pus and blood in urine	4		
Tubercle bacilli on culture (2 were positive in 1955 before chemotherapy)	0	15	15
Intravenous pyelogram abnormalities	9	2	11

Some indication of the degree of renal involvement by tuberculosis is given by the pyelograms reproduced in *Figs.* 513–516 and the accompanying case summaries, which may be considered representative of the group of survivors.

DISCUSSION

The results of this survey on long-term survival of patients with renal tuberculosis not treated by nephrectomy or chemotherapy vindicate the view advanced in 1929 that patients with small renal lesions do not invariably progress to complete destruction of the kidney, but may survive. Many of these patients continued to have active renal tuberculosis for many years, but without marked destruction of the kidney, while some became free from tubercle bacilli and free from pus and blood in the urine.

The present series is of value in indicating the natural history of untreated renal tuberculosis as a basis for comparison with the recently adopted conservative drug treatment of this disease. It suggests that nephrectomy for tuberculosis was not an emergency procedure, even before the days of chemotherapy, and to-day it should be reserved for those kidneys which do not respond to chemotherapy in a reasonable time.

SUMMARY

Eighty-two cases of renal tuberculosis not treated by nephrectomy or anti-tuberculous drugs have been followed for 20 to 35 years. Of these, 61 had bone and joint as well as renal tuberculosis. Twenty-six patients are alive and well and 5 died after surviving 20 or more years, a 20-year survival of almost 38 per cent. Nineteen patients died between 5 and 20 years after diagnosis, 31 patients died within 5 years of diagnosis, and 1 case was lost to follow-up in the first year.

Of the living group of 26 patients, there are recent pyelograms in 15, of which 11 are abnormal.

There were 55 deaths; 22 patients died of renal tuberculosis, 26 of non-renal tuberculosis, and 7 of non-tuberculous causes. Thus in a 20- to 35-year follow-up of 82 cases of renal tuberculosis, 26·8 per cent died of this disease, compared with 38 per cent who survived for over 20 years.

Acknowledgements.—The authors are grateful to Dr. C. A. Wicks, Superintendent, Toronto Hospital for Tuberculosis, for permission to conduct this study; and to Dr. J. C. McClelland, the surgeon in charge of the Urological Service at the Toronto Hospital for Tuberculosis since 1930.

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PRIMARY FIBROSARCOMA OF THE TONGUE ASSOCIATED WITH SYPHILITIC LEUCOPLAKIA

By R. A. CAWSON, London

Fibrosarcoma of the tongue is an uncommon condition which may rarely occur as a sequel to irradiation. The case described here, which presented a difficult diagnostic problem, was originally thought to be a case of irradiation-induced sarcoma.

CASE REPORT

In 1946 the patient, a woman of 47 years, complained of soreness of the tongue. On examination there was leucoplakia of the tongue with some raw areas, and the Wassermann reaction was positive. In spite of several courses of penicillin and bismuth, the Wassermann reaction remained positive, and in 1951 the patient complained of a lump on the tongue. The tongue showed areas of leucoplakia on the dorsum, and on the posterior third a lump which was diagnosed clinically as a carcinoma.

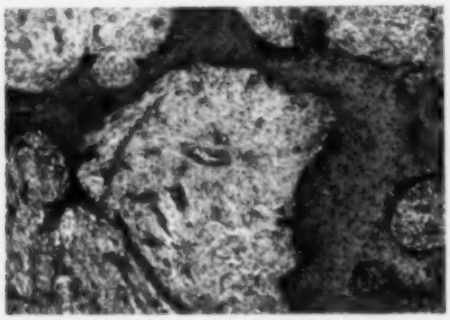

Fig. 517.—The first biopsy, showing epithelial hyperplasia.

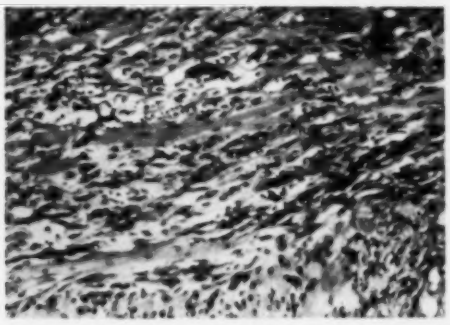

Fig. 519.—The final biopsy, taken just before excision of the tongue, showing infiltration of muscle.

In addition, the patient had had increasing shortness of breath for the past year, and radiographs of the chest showed appearances suggestive of syphilitic aortitis.

The patient was referred for radiotherapy, and the report at that time, on a biopsy taken prior to treatment (*Fig.* 517), was of a well-differentiated squamous-cell carcinoma with associated inflammatory changes.

Two courses of radiotherapy were given by means of the radium beam and radium needles, and a final biopsy taken in April, 1952, stated that no residual carcinoma cells were present (*Fig.* 518). Clinically, however, an ulcerated lump was still present on the posterior part of the tongue so that in July the patient was referred back for further surgical treatment.

Biopsy of the mass at this time was thought to show no clear evidence of malignancy, and a local excision was carried out. The mass, however, recurred, necessitating two further excisions, though the histological picture of these biopsies showed no change. A deeper biopsy taken in January, 1953, showed a fibrosarcoma infiltrating muscle (*Fig.* 519).

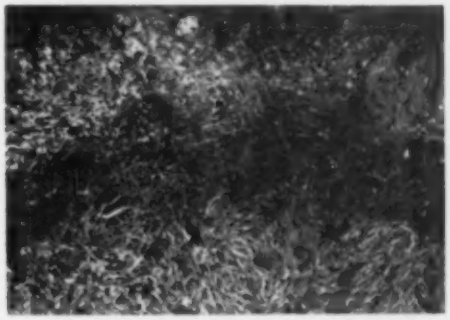

Fig. 518.—The biopsy taken immediately after completion of irradiation showing the appearances of a fibrosarcoma.

At this time the patient appeared well, had no pain, but had some difficulty in swallowing, and occasional small hæmorrhages from the growth. The mass on the tongue was predominantly on the left side, and was rounded, lobulated, and overlapped the normal surface of the tongue at its margins. The posterior limit of the mass could not be felt. There were no lymph-nodes palpable, but there was a fullness in the submandibular region, and the underlying tissues felt firm and thickened. (*Fig.* 520.)

Total glossectomy was carried out, and the patient recovered satisfactorily. (*Figs.* 521, 522.) She was discharged six weeks later with the wounds almost healed. A month later, however, though she was comfortable and was swallowing well, a small lump was noticed near the epiglottis. Thereafter there was increasing pain and a growing mass which became ulcerated and fixed to the floor of the mouth and mandible, causing progressive dysphagia and dyspnœa. The patient died six months after her last operation, that is, seventeen months after the radiotherapy for the original mass had been completed.

At post-mortem examination the growth was found to consist of a lobulated swelling filling the floor of the mouth, overhanging and partially obstructing the glottis posteriorly. The regional lymph-nodes were normal and there was no evidence, naked-eye or histological, of metastases in the lungs or elsewhere. Section of the aorta showed the histological picture of syphilitic aortitis.

DISCUSSION

This case presented a difficult problem in diagnosis. The mass on the tongue had been reported on initially as a carcinoma and finally as a sarcoma. In the period between these reports, and following

irradiation, the mass had continued to grow in spite of excisions, but no firm diagnosis was obtained until there was clear histological evidence of infiltration of muscle. The diagnosis of fibrosarcoma was confirmed at the post-mortem examination. It is also certain, however, that the patient had had syphilis and leucoplakia of the tongue for a number of years, and the association of these conditions with carcinoma is widely accepted, though this, of course, does not exclude the possibility of another type of neoplasm occurring in the adjacent tissues.

FIG. 520.—The tongue, approximately a year after the first biopsy, showing the lobulated mass of the neoplasm posteriorly and the extensive leucoplakia anteriorly.

FIG. 521.—The tongue excised at operation, showing the dorsal surface.

At first sight, then, it would appear that this was a case of carcinoma of the tongue followed by an irradiation-induced fibrosarcoma.

It is reasonable, therefore, to compare the present case with other such cases reported previously. The development of fibrosarcoma of the tongue following irradiation has been reported on eight occasions.

Gricouroff (1937) reported 2 cases; in the first case sarcomatous change followed twelve years, and the second four years, after radiotherapy. Gricouroff (1943) also reported another case of sarcoma of the tongue occurring four years after irradiation. Deller (1951) describes a fibrosarcoma appearing nineteen years after radiotherapy for a growth of which the histology is not known. In the cases described by Marqués, Planel, and Hermous (1952) the latent period was four years, and in the case described by Gilbert, Wallon, and Andrieux (1952) the latent period was fifteen years. Durden-Smith and Weavers (1953) describe a similar case following nine years after irradiation, and Thomas and Bonneau (1955) describe a fibrosarcoma of the tongue following three years after irradiation of a carcinoma of the pharynx.

In most of these cases the lesion for which irradiation was given was a squamous-cell carcinoma of the tongue. The exceptions are one of Gricouroff's cases, Thomas and Bonneau's case, where the original growth was in the pharynx, and Deller's case, where the histological diagnosis was unknown.

Willis (1953) states that when neoplastic change follows the use of radio-active substances (rather than X rays) the change is usually sarcomatous and only rarely carcinomatous. Further, sarcomatous change is more likely when the maximum effect of the irradiation occurs deeply in the tissues rather than through the surface. Both of these conditions are fulfilled when growths of the tongue are treated by the use of interstitial radium needles, and this was the method employed in all the cases mentioned except one. In this case, described by Thomas and Bonneau (1955), a carcinoma of the pharynx was treated by means of X rays, and this was followed three years later both by a sarcoma of the tongue and by a carcinoma of the palate. The present case, which was treated with the radium beam and then with radium needles, has features in common with those just described, but differs from them in the extreme brevity of the latent interval. Thus, the biopsy which showed clear evidence of infiltration of muscle by a sarcoma was taken less than a year after radiotherapy had been completed.

FIG. 522.—Sagittal section of the tongue, showing the pale homogeneous appearance of the cut surface of the neoplasm in its posterior part.

There seemed to be three possibilities as to the nature of this case. First, the lesion might, in fact, be a fibrosarcoma following irradiation, but with unprecedented rapidity. Secondly, there might have been a coexisting carcinoma and sarcoma, but that

in the original biopsy of superficial tissue the appearance of the stroma was misleading. Thirdly, this case might have been one of a fibrosarcoma associated with pseudo-epitheliomatous hyperplasia. The earlier sections were therefore reviewed with these possibilities in mind, and in the light of a knowledge of the final nature of the condition.

The original biopsy is of a small fragment of tissue and shows strikingly hyperplastic squamous epithelium and cellular, pleomorphic connective tissue. There are inflammatory changes more severe in some parts of the section than in others. The nature of the epithelial hyperplasia must, however, be considered in relation to non-specific inflammatory changes on the surface and in relation to the long-standing syphilis and leucoplakia. In addition, if sarcomatous change had already occurred in the connective tissue this might also have affected the epithelium. Thus, pseudo-epitheliomatous changes in the overlying epithelium are highly characteristic of a granular-cell 'myoblastoma' (Azzopardi, 1956), and though it is not suggested that the lesion in the present case is in any way related to a 'myoblastoma', yet it is possible that an analogous change has occurred in the epithelium. The final conclusion reached from examination of this section is, in fact, that the epithelial changes are not sufficient to justify the diagnosis of carcinoma.

With regard to changes in the connective tissue, though it is unquestionably abnormal in the original sections, it is probably not possible to make a diagnosis on this small superficial fragment. Sections taken immediately upon completion of radiotherapy, however, show the appearances of a fibrosarcoma. An important diagnostic difficulty after irradiation is the occurrence of irradiation fibromatosis as described by Pettit, Chamness, and Ackermann (1954), who believe that it may be impossible in some cases to distinguish between irradiation fibromatosis and fibrosarcoma unless there is clear evidence of infiltration. In the present case, evidence of infiltration was unfortunately only obtained in the later sections. The histological appearances of the various specimens, including that taken immediately after irradiation, are those of a fibrosarcoma. Since, in addition, there was a growing mass on the tongue before irradiation was started, and since the evidence for the presence of a carcinoma is inadequate, it must be concluded that this is a case of primary fibrosarcoma of the tongue.

The incidence of primary fibrosarcoma of the tongue is not easy to determine, as in many of the earlier reports the diagnosis would not now be substantiated. Thus, Fripp and Swan (1902), in a review of 43 cases reported as sarcoma of the tongue, found that 29 were acceptable, and that of these only 4, including their own, could be described as fibrosarcomata. In a review by Coughlin (1915) 58 cases are discussed, but none of these appears to be fibrosarcomata. More recently Koerth, McCorckle, and Donaldson (1943), in personal communications with Ewing and Wirth of the National Cancer Institute, found that neither of these workers had in their extensive records a single example of this condition. Further, this condition does not appear in the analysis of 2000 human neoplasms by Lillie (1941). Koerth and others considered, therefore, that primary fibrosarcoma of the tongue was one of the rarest neoplasms, and Hofman, Martin, and Marti (1947) consider that only about 20 cases have been reported since 1867.

It is apparent, therefore, that radiation, particularly by means of interstitial needling, is an important cause of fibrosarcoma of the tongue. Gricouroff (1943) found that of 1700 biopsies of the tongue there were 5 specimens of fibrosarcoma, but 3 of these were cases following irradiation that he had reported.

In so far as any conclusions can be drawn regarding the clinical features of these rare tumours, the present case appears largely to conform with cases previously reported. The unusual features of this case, however, were the difficulties of diagnosis and the association with persistent syphilitic leucoplakia. This association, which must be presumed to be quite fortuitous, is not mentioned in any of the 29 cases of all types of sarcoma of the tongue reviewed by Fripp and Swan (1902), and is not considered by Coughlin (1915), Koerth and others (1943), Ulin, Shoemaker, and Froio (1954), or Hofman and others (1947) in their reviews.

With regard to the management of this condition, it is generally agreed that surgery is the treatment of choice, as this neoplasm is radio-resistant. Pettit and others (1954) consider that fibrosarcomata following irradiation show less tendency to metastasize and to cause death by local extension. The tumour in the present case, however, also showed this mode of behaviour.

SUMMARY

1. A case of primary fibrosarcoma of the tongue is described.

2. This sarcoma was associated with syphilitic leucoplakia of six years' duration.

3. The diagnosis of fibrosarcoma was made only after the patient had received radium therapy, but reasons are given for the conclusion that this neoplasm preceded irradiation.

4. The conditions of primary fibrosarcoma of the tongue and fibrosarcoma following irradiation are briefly reviewed, and it would appear that the irradiation-induced variety is no more uncommon than the other.

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SUBMUCOSAL VASCULAR PATTERNS OF THE ALIMENTARY TRACT AND THEIR SIGNIFICANCE

BY P. S. BOULTER AND A. G. PARKS, LONDON

As the scope of intestinal surgery expanded, it was realized that the presence of anastomotic channels is of great importance in maintaining an adequate blood-supply to suture lines. The ramifications of vessels approaching viscera have therefore been intensely studied. Physiologists have shown particular interest in the small vessels within the walls of the stomach and intestine, especially those in the mucosa which play so vital a part in absorptive processes. Their histological details have been known for many years, but less attention has been paid to the patterns of distribution of intramural vessels.

In America, Noer and Manning (1942) and Noer and Derr (1949) showed the ramifications of vessels in the small intestinal wall by injection of coloured media and did much to clarify their anatomy. Later research by Ross (1950) demonstrated anatomical variation between the large and small intestine. His work was based upon the intravascular injection of celloidin and subsequent digestion of surrounding soft tissues. A framework of vessels was left which could only be studied divorced from the tissues which they supplied. These preparations gave valuable information about such features as anastomoses in the antimesenteric wall of the bowel, but do not help to clarify the constitution of finer vascular plexuses.

The development of microradiology stimulated further interesting work on vascular patterns. The study reported by Barclay and Bentley (1949) on the gastric vessels pointed the way to more research, particularly that on arteriovenous shunts. Microradiology, however, has major limitations, as all that can be seen is a two-dimensional pattern of vessels without detail of the surrounding tissues. Later work by Barlow and Bentley and their colleagues (1951, 1952) extended earlier techniques by combined radiological and histological studies. Their preparations were mostly made by injection and subsequent microdissection and have shed new light on intramural vessels of the stomach. An interesting feature of their work has been anatomical proof of the existence of arteriovenous communications in the gastric wall—a subject that has been a matter of controversy.

Their investigation was the initial stimulus to the present study, which had two main objects. These were:—

1. To attempt further demonstration of intramural vessels in various parts of the alimentary canal.

2. To clarify some of the uncertainties relating to the presence and nature of arteriovenous communications.

Our attention has been focused on the vessels of the submucosal plexus which is the richest vascular network of the gastric and intestinal wall. Surgical attention is drawn to these vessels because of the part that they are likely to play in the healing of anastomoses and in the genesis and repair of inflammatory lesions.

MATERIAL AND SOURCES

Whenever possible this study has been carried out on fresh human viscera.

The decision to use fresh tissue was made when it was realized that post-mortem material had several drawbacks. These included: (1) difficulty in washing out all corpuscles from vessels prior to injection, and (2) the severe degree of mucosal autolysis which rendered dissection of the submucosa virtually impossible. We have therefore relied almost entirely upon specimens obtained at partial gastrectomy, hemicolectomy, abdominoperineal excision of the rectum, and other similar operations.

Because of the scope of present-day surgical practice, the selection of viscera available was not as complete as could be wished. Specimens of upper small bowel were obtained with difficulty owing to the relatively infrequent resection of significant lengths. There was no problem at all in getting sufficient partial gastrectomy specimens removed at operation for duodenal ulcer. Some parts of stomach and duodenum were obtained at partial and total pancreatectomy, while terminal ileum was obtained at right hemicolectomy. Specimens of normal and abnormal cæcum, colon, and rectum have been plentiful. Altogether 45 stomachs, 16 segments of colon and rectum, and 12 segments of small bowel have been studied.

METHODS

Within an hour of removal of specimens the largest available artery and vein were cannulated. In some instances several vessels were used in order to produce adequate vascular filling of longer segments of bowel. It was found advisable to ligate all surplus vessels to prevent leakage. This also avoided contamination and staining of visceral surfaces.

The ideal material for cannulæ is polyethylene (polythene) tubing which can be adapted to any

syringe or needle. The cannulæ themselves require little preparation. A flange can be formed at each end of the required length of tubing by cautious warming near a match or Bunsen burner flame. This serves two useful purposes. First, it facilitates the fixation of the cannulæ, which can be tied in place by one fine silk ligature. Secondly, an extra buttress of polyethylene is built up at the syringe or needle end, which prevents splitting of the material and consequent leakage.

When the cannula is securely in place all corpuscles are washed out of the vessels. It was found that this was best achieved with normal saline injected via the arteries by a syringe or a simple pressure perfusion apparatus. Whichever method is used, it is important to avoid the introduction of air-bubbles which can ruin a preparation by preventing exsanguination and blocking the infusion of dye into large segments of tissue.

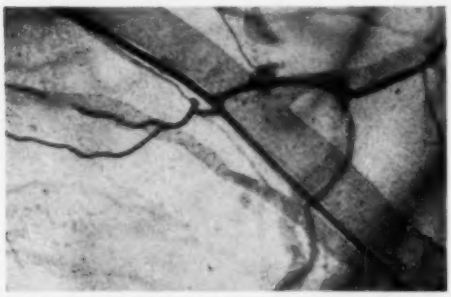


FIG. 523.—Vessels in the gastric submucosa stained by intravascular hæmatoxylin. Differentiation between arteries and veins is easy.

FIG. 524.—The submucosa of a partial gastrectomy specimen. The vessels are outlined by silver.

It was at this stage that an indication of the richness of vascular patterns was often given. An entire stomach cleared of corpuscles could be filled with dye through a single vessel such as the left gastric or one of the gastro-epiploic arteries. Similarly, a 2 ft. 6 in. length of colon has been seen to leak perfused fluid from one cut end when injection was being made into a vessel at the other. This was only partly due to the presence of the marginal vessels.

Various injection methods and media have been used. Occasionally, when fresh tissue was injected, some method of quelling turbulently contracting bowel had to be used. Intra-arterial 1 per cent procaine adequately relaxed both bowel musculature and the walls of intrinsic vessels. This often added considerably to the ease of injection.

An assortment of material was used for display of vessels, all of which were introduced by intravascular injection. These included: (1) Indian ink. (2) Gelatin injection masses with added dyes (Barlow, 1951–1952; Barlow, Bentley, and Walder, 1951; Barlow and Bentley, 1951). (3) Neoprene latex. (4) Pigmented dispersion media (Millen and Woollam, 1953; Woollam and Millen, 1955). (5) Dyes, e.g., hæmatoxylin, anthracene blue, methyl red. (6) Silver solutions.

No single material was found to be absolutely satisfactory. We found that the most generally useful preparations were those obtained by intravascular staining with dyestuffs or silver.

It was found after considerable trial of dyes that there was none as consistently satisfactory as hæmatoxylin (*Fig.* 523), which was found to be a good vascular stain by Grant (1930) and Grant and Bland (1931).

After washing the specimens the vessel walls were fixed by perfusion for at least one hour with warm 10 per cent formol saline. After some had run through, a pressure of 60–100 mm. Hg was maintained by clipping the venous cannulæ. Normal saline was then perfused to displace the formalin and Delafield's hæmatoxylin was injected until it flowed freely from the venous cannulæ. These were again clipped and a pressure of 80 mm. Hg was maintained. This intravascular staining was continued for 2 to 4 hours, and after that time the hæmatoxylin was washed out of the vessels by perfusion with tap water, thus 'blueing' the dye.

Satisfactory differential staining of arteries and veins was obtained (*Fig.* 523). It was interesting to note how very little extravascular diffusion of the dye occurred. In this aspect hæmatoxylin scored over all other dyes that were tried.

Staining with intravascular silver was used on a number of occasions and proved reasonably satisfactory. Good and often dramatic arteriovenous differentiation was given (Boulter and Parks, 1951).

After washing, a 1/200 solution of silver nitrate was introduced through the arterial cannulæ and the tissue was perfused. Pressure was maintained, as in the hæmatoxylin method, but for a very much shorter time (15 minutes). After this the excess silver was washed out with tap water. Silver, together with the chloride in the vessels at the beginning of perfusion, produced a deposit of silver chloride in the vessel lumen and wall. Subsequent reduction to metallic silver was achieved by perfusion of a 1/30 solution of KD 76 photographic developer for 10 minutes, followed by further washing and formalin fixation.

Both methods were frequently used, most of the preparations that were eventually studied being produced by one or other techniques.

Three types of preparation were studied. Thick celloidin sections were prepared by a method (Parks, 1956) developed from the work of Chesterman and Leach (1949). This proved to be a very simple

technique for making serial sections. Clear views of vessels are obtained and the process of preparation and cutting is easily mastered.

Frozen sections were also made, mostly of 100–200 μ thickness. Most of these were not, in fact, intended as permanent preparations, but were designed to check the adequacy of staining of aliquot portions of large specimens.

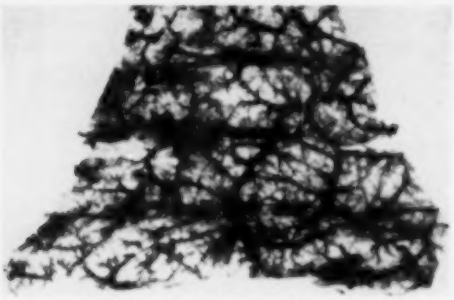


FIG. 525.—Gastric submucosa from the anterior wall of the pyloric antrum. Intravascular hæmatoxylin.

Most of our preparations, however, were prepared by selective stripping of the muscular and mucosal layers, leaving behind the delicate submucosal layer with all its vessels.

The viscus was opened in a suitable line and cut into pieces. These were numbered and labelled for subsequent recognition and the pieces pinned out flat on a cork board. Injection of formol saline into the submucosa made the removal of the muscular layer relatively easy. The muscle was usually

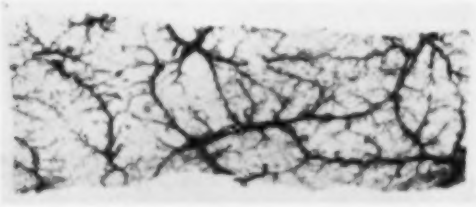


FIG. 527.—Submucosa of transverse colon. Intravascular hæmatoxylin.

discarded and the remainder of the specimen fixed in formol saline. After two days it was dehydrated by immersion in increasing concentrations of alcohol. The final twenty-four hours of impregnation in absolute alcohol rendered the mucosa brittle and it was peeled off, leaving the submucosal layer with its vessel plexus intact and unobscured. This process requires some delicacy lest there be undue tearing of the submucosal plexus.

These sheets of vessels with a surrounding frail stroma were then cleared in xylene and stored in cedar-wood oil or a neutral mounting medium. The ideal method of storage was by mounting the specimens between slides of plate glass cut to the required size. The sizes of the preparations varied from postage-stamp portions to the entire submucosa of a partial gastrectomy specimen stripped in one piece (*Fig.* 524).

This method was satisfactory for stomach, ileum, colon, and rectum. Unfortunately, it was not very successful in duodenum and upper jejunum. Close adherence of glandular tissue to the muscle in the crypts of Lieberkuhn and below the valvulæ conniventes rendered difficult any separation through this tissue plane.

OBSERVATIONS

Inspection and comparison of plexuses seen in the stripped preparations from various parts of the gastro-intestinal tract showed a striking variation in appearance and richness of the submucosal vessels. There

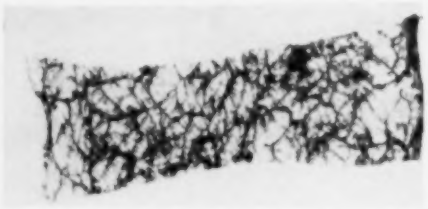


FIG. 526.—Submucosa of terminal ileum. Intravascular hæmatoxylin.

was a remarkable specificity of pattern from organ to organ in various parts of the bowel wall. Typical gastric, duodenal, jejunal, ileal, and colonic patterns were identified. In the stomach a rich plexus of large and small vessels was found with extensive interarterial and arteriovenous anastomosis (*Fig.* 525).

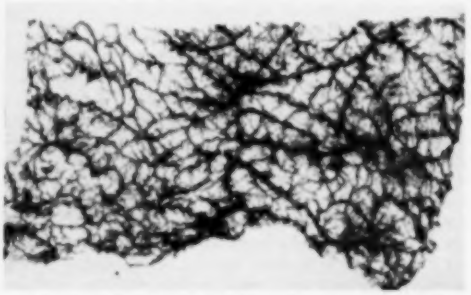


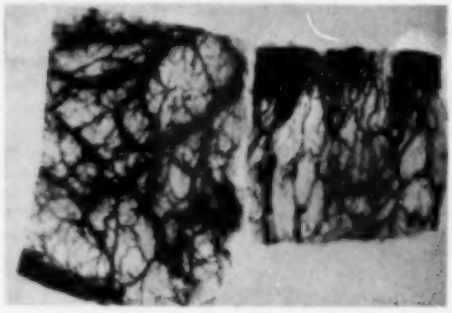
FIG. 528.—Submucosa of distal sigmoid colon. Intravascular hæmatoxylin.

Large vessels were seen in the submucosa up to 2 mm. in diameter and an extensive network of smaller ones filled the interstices of the pattern almost completely. The venous lagoons noted by Barlow (1951) were observed, and it was noted that these were sometimes related to the mouths of arteriovenous communications.

The small bowel has a characteristic fine mesh plexus with numerous vessels of up to 0·3 mm. diameter (*Fig.* 526). Innumerable interarterial anastomoses occur, particularly in the middle and terminal ileum. The pattern in the first part of the duodenum is quite different from that in other sites, being marked by numerous longitudinal vessels with fewer cross-communications.

The colonic pattern differs in its detail at various levels. In the cæcum, ascending transverse and descending colon there is a coarse meshwork of rather

larger vessels (up to 0·6 mm.) with relatively few cross-communications, and large vessel-free spaces (*Fig.* 527). In the distal sigmoid colon the appearance of the plexus then changes again and a more lavish vascular supply is evident (*Fig.* 528). There are still the large vessels noted in the rest of the colon, but



A B

FIG. 529.—Rectal submucosa. A, Upper rectum. B, Just above anorectal ring. Intravascular hæmatoxylin.

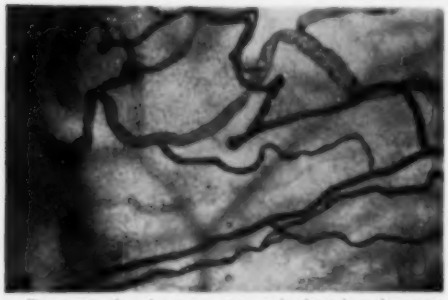


FIG. 531.—Arteriovenous communications in submucosa of greater curve of stomach. Diameter of narrowest part of loop is 33μ. Intravascular hæmatoxylin.

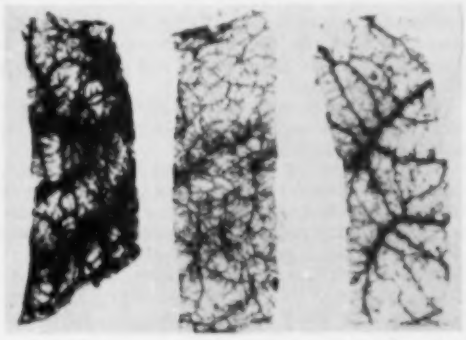
superimposed on this arrangement there is a series of smaller vessels with rich anastomosis reminiscent of the small bowel pattern. It is tempting to relate this increased richness to the poorer mesenteric anastomoses of the sigmoid. As the rectosigmoid junction is approached the concentration of vessels increases, and in the rectum there is a rich meshwork of vessels of all sizes reminiscent of the gastric pattern (*Fig.* 529), but with the main channels running longitudinally. It is in this plexus that the branches of the superior hæmorrhoidal artery terminate.

As shown in *Fig.* 530, there are three strips of mucosa from stomach, small and large intestine. This demonstrates the marked difference in appearance of the plexuses in these areas.

In the stomach, examination of thick celloidin serial sections and of stripped preparations showed a large number of loops of greater than capillary size interposed between arteries and veins. These arteriovenous communications were mostly 30–35 μ in diameter and were found with equal frequency on the greater and lesser curvatures. These were all of a simple loop form which passed with almost even calibre from artery to vein (*Fig.* 531).

Similar communications of this type were also seen in the small bowel (*Fig.* 532), colon, and rectum, but with very much less frequency.

Attempts were made to count the numbers of shunts in given segments of tissue, but it is hard to



A B C

FIG. 530.—Three strips of submucosa for comparison. A, Posterior wall of stomach. B, Mid-ileum. C, Cæcum (note resemblance to transverse colon in *Fig.* 527).

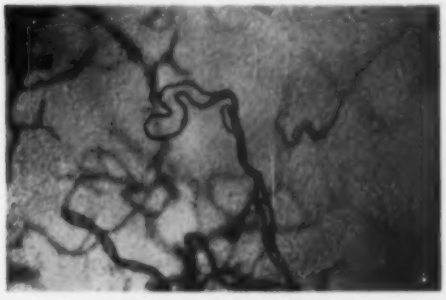


FIG. 532.—Arteriovenous loop in ileal wall—diameter is 30μ. Ghosts of other vessels are seen out of focus. Intravascular hæmatoxylin.

give a tally of any accuracy because of the difficulty of counting in these thick preparations. As an indication of the relative richness of communication, twelve shunts have been found in 1 sq. cm. of whole gastric submucosal preparation, while in a piece of rectal wall only two were seen. Segments of colonic wall have been examined with similar findings.

DISCUSSION

The arrangement of intramural gastric vessels accorded with the description of Barlow, Bentley, and Walder (1951). No specialized arteriovenous anastomoses were seen with the 'Quellzellen' structure reported in the skin by Grant and Bland (1931) and the ear and tongue by Prichard and Daniel (1956). Some more complicated arteriovenous anastomoses were seen in mesenteric preparations and these are being further investigated. Repeated attempts were made to find the complicated type of glomus structure reported in the gastric wall by De Busscher (1948), but these were not seen, and none of the complicated networks of arteriovenous anastomoses described by Clark (1939) and Clara (1938) was found. It seems probable that the

majority of the loop-like communications in the submucosal plexus are channels of an expanded capillary type which have been described in the mesenteries by Chambers and Zweifach (1944) and, more recently, in the bulbar conjunctiva by Spence Meighan (1956). It would be interesting to note whether spontaneous vasomotion occurs in these vessels, but highly refined techniques would be necessary for such observations and these have not so far been mastered.

There are three main aspects of the present work which are of particular clinical interest and which merit further research.

The presence of arteriovenous communications in equal profusion on both curvatures of the stomach would suggest that they are not *per se* likely to play the important part in disease that has been postulated. Additional factors must be sought to explain the selective occurrence of benign peptic ulcers on the lesser curvature. It was repeatedly noted while preparing gastric specimens that the submucosa of the lesser curve was tethered to muscle, whereas in other parts it could be separated by as much as 2 cm. by injection of saline. This adherence to the muscular layer was due mainly to the direction of entry of the arteries to the lesser curve, which as Barlow (1951) showed, passes straight to the mucosa with minimal submucosal ramification. The tethering of duodenal mucosa to the underlying muscle has already been noted.

The demonstration of morphological variation in the submucosal plexuses of different organs adds some information to that already held. There seems to be some relationship between the richness of the submucosal plexus in a segment of bowel and the security of healing of an anastomosis performed in it. Further work is in progress in an attempt to provide experimental evidence in support of this observation.

The vessels which must carry blood to wounds in the intestine are those of the submucosal and subserosal plexuses, of which the former is the larger and more important. It is possible to speculate on various methods of suture that could minimize damage to the vessels and consequent infarction of the bowel ends used in anastomoses.

In the physiological study of exercise and shock the marked variation in splanchnic blood-flow (Wade, Coombs, Childs, Wheeler, Cournard, and Bradley, 1956; Reynell, Marks, Chidsey, and Bradley, 1955) is of interest. The great capacity for shunting blood from arterial to venous sides through arteriovenous communications in the viscera and their mesenteries may be significant.

Relatively small alterations in the diameter of participating vessels would lead to considerable alteration in flow, as would be predictable by Pouseille's law which relates flow to the fourth power of the radius of the tubes concerned. This could account for sudden increase in the return of blood to the right side of the heart in exercise and shock.

SUMMARY

The observations made in a series of preparations of vessels in the stomach and bowel walls are described. Certain points of possible significance are discussed.

Acknowledgements.—The authors owe their sincere thanks to Mr. H. J. B. Atkins, in whose department most of this work has been done, and to the other surgeons of Guy's Hospital for their generous encouragement and help. Part of this work was done while one of us (P. S. B.) held a research grant from the Endowments Committee of Guy's Hospital, to whom we express our gratitude. We thank Miss R. M. Hills for her untiring technical help.

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EXPERIMENTAL SURGERY

THE ABSORPTION OF SURGICAL CATGUT

II. THE INFLUENCE OF SIZE

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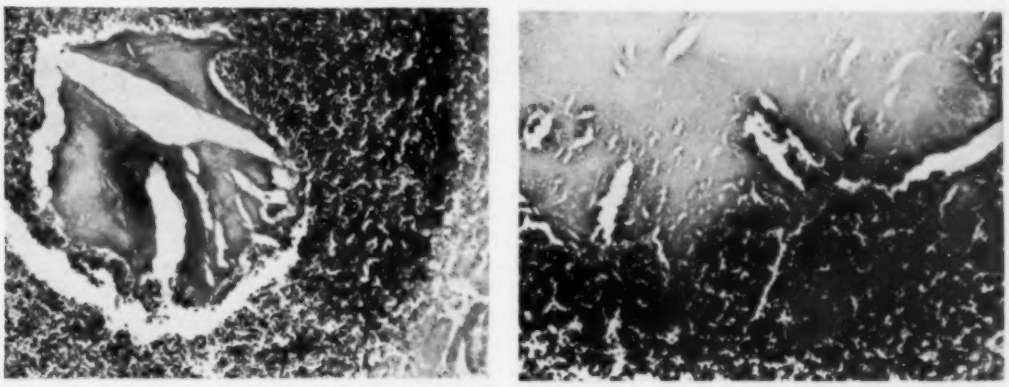
In our previous paper (Lawrie, Angus, and Reese, 1959) we described the fate of fine catgut which had been implanted in the lumbar muscles of the rat. In these experiments plain and medium chromic catgut of sizes 3/0 and 5/0 were used. Plain catgut evoked a brisk pyogenic reaction which in 3 days produced a sterile abscess around the suture. The pus digested the catgut, the tensile strength of which diminished rapidly. By contrast, medium chromic catgut produced a very slight inflammatory reaction and was absorbed purely by foreign-body giant cells which began their attack 10 days after implantation.



A B

Fig. 533.—Plain catgut 3 days after implantation. A, Size 0. An abscess several times the diameter of the suture. Half the suture has been digested away. B, Size 2. An abscess which is beginning to digest the surface of the suture. H. and E. (× 120.)

As an absorbable surgical suture, Harvey (1934), Bates (1939), Mahoney (1943), and Pickrell and Clay (1944) favoured fine chromic catgut. In their experience this caused less tissue reaction and was less rapidly weakened than either plain gut or chromic gut in the larger sizes.

We have now implanted sizes 0 and 2 of both plain and medium chromic catgut in the lumbar muscle of the rat under the same conditions as before and have measured the tensile strength and examined the histological appearances at varying intervals after implantation.

MATERIALS AND METHODS

Wistar albino rats of average weight 160 g. were used. They were anæsthetized with ether, and under aseptic conditions a midline dorsal incision was made in the skin. For histological examination the implantations were made twice on each side through exposed lumbar muscle with a 45-mm. curved intestinal needle. A total of 304 rats were used. At varying intervals the lumbar muscles were excised and fixed in 10 per cent formol saline. Blocks were embedded in paraffin and transverse sections were cut at 6 μ and stained with hæmatoxylin and eosin.

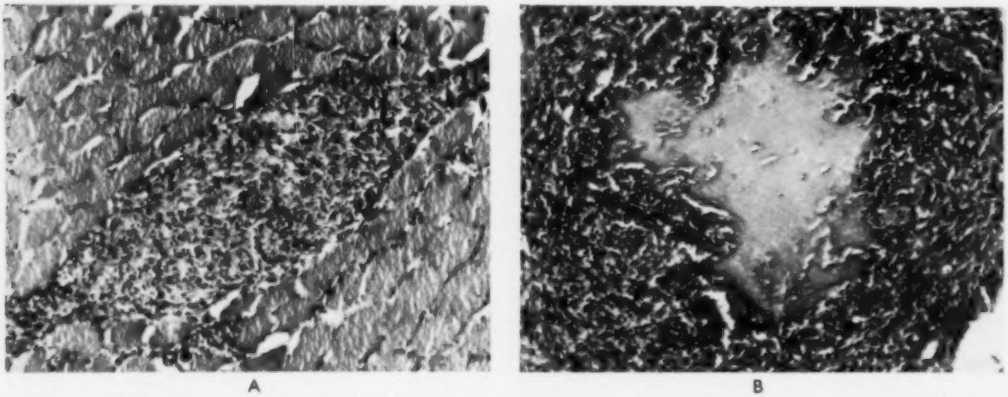
For examination of tensile strength, the catgut was first passed through a polythene tube having a window 1 cm. long cut out of one side. Polythene tubes and catgut were then implanted in the lumbar muscles, two on each side of the rat. A total of 165 rats were used. At the times required, the implants were withdrawn and tested on an inclined plane tensiometer. The methods are given in greater detail by Lawrie (1959) and Lawrie and others (1959).

RESULTS

Histology.—

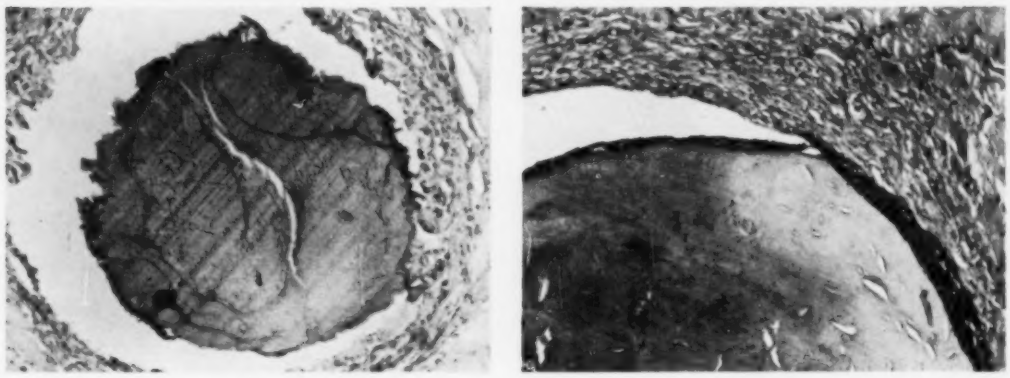
Size 0 Plain Catgut.—At 24 hours the suture was still intact. It was covered by a layer of polymorphonuclears and some blue-staining stringy debris formed from degenerate polymorph nuclei. The muscle around the suture showed mild inflammatory changes. By 2 days every suture had been attacked, usually with digestion of a quarter of the area of

cross-section. Digestion of half the area was not uncommon, and in one case only fragments were left, lying in a pool of pus. In 3 days the abscess had grown often to six times the diameter of the suture (*Fig.* 533 A). The pus was divided into two zones, an inner one of fresh polymorphs and an outer one of blue stringy material. Granulations lining the abscess were only muscle. The suture was intact and covered with a layer of polymorphs and a little blue stringy material. In 3 days less than a quarter of the suture had been digested (*Fig.* 533 B). The abscess did not reach a maximum until 4 days, by which time about half the area of cross-section of the suture had gone. Only a single suture showed total digestion at this stage, but



A B

FIG. 534.—Plain catgut 13 days after implantation. A, Size 0. No suture remains, only a small clump of granulations. B, Size 2. A half-digested suture which still has pus cells in contact with it, but it is also being attacked by giant cells. H. and E. (× 120.)



A B

FIG. 535.—Medium chromic catgut 12 days after implantation. A, Size 0; B, Size 2. Quiet narrow granulations with superficial attack on the suture. H. and E. (× 120.)

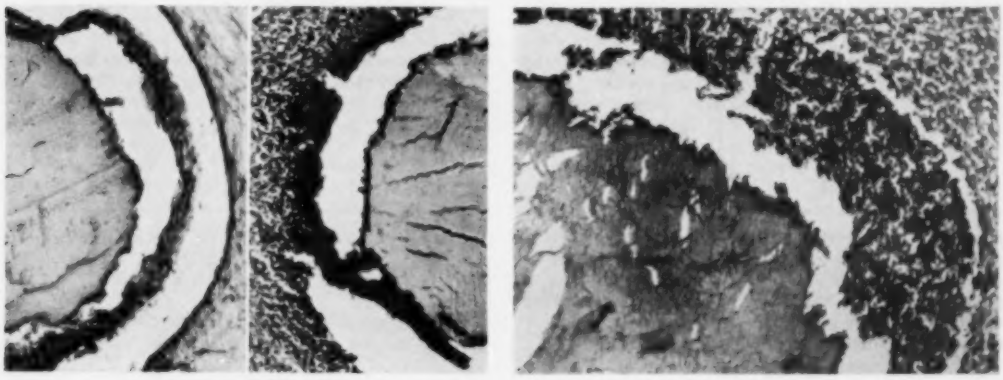
just forming and the pus had extended in some places into the surrounding muscle where there was more inflammation than before. A number of sutures had by now disappeared altogether; in most of those that survived, half the area of cross-section was gone. By 4 days the abscess had shrunken somewhat and was surrounded by a layer of active granulations. Most of the sutures had been completely digested by 5 days, but a few persisted until 10 days, when all the pus had absorbed and giant cells surrounded the suture remnants. After this, suture material was scanty and engulfed by giant cells. At 15 days a clump of macrophages the diameter of the suture was present (*Fig.* 534 A) and at 25 days a thin scar.

Size 2 *Plain Catgut.*—Sections 24 hours after implantation showed the usual inflammation of the by 7 days half of them had completely dissolved, the survivors being reduced to a quarter their former size. At 10 days, if suture was still present, it was surrounded by pus. When no suture remained, the pus was completely absorbed and there was just a mass of granulation tissue twice the diameter of the suture. The picture at 13 days was similar, with rather less pus, but giant cells were also attacking the suture remnants (*Fig.* 534 B). These had all gone by 15 days and at 25 days there was nothing but a narrow scar.

Size 0 *Medium Chromic Catgut.*—The first 5 days showed the development and disappearance of the mild inflammatory reaction in the surrounding muscle, and the formation of a band of granulations around the suture. During this time the suture itself

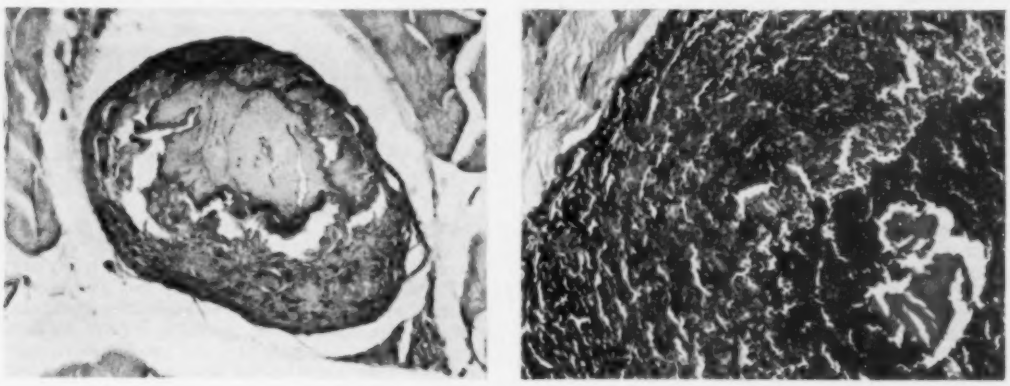
was intact, and covered with a very few polymorphs or a little blue debris. From 7 days the granulations were narrow and condensed, but there was no attack on the suture until 12 days, when a few giant cells appeared (*Fig.* 535 A). At 13 days many of the sutures were bathed in pus, which was digesting them.

Size 2 Medium Chromic Catgut.—The mild inflammatory reaction present at 24 hours increased in intensity in the first few days. At 3 days granulations had formed around the suture. These became thicker and more active until 7 days, when the lesion was about twice the diameter of the suture. For the first



A B

FIG. 536.—Medium chromic catgut 15 days after implantation. A, Size 0. Two sutures lying adjacent in the same animal. A quiet lesion with superficial attack and a purulent lesion. B, Size 2. A lesion with thick granulations and free pus. The suture is reduced by about one-third. H. and E. (× 120.)



A B

FIG. 537.—Medium chromic catgut 35 days after implantation. A, Size 0. A quiet granuloma. Giant cells are removing the suture, which is reduced by about half. B, Size 2. An active lesion with the last remnants of the suture being digested by fresh pus. H. and E. (× 120.)

When this occurred, the surrounding granulations were much more active. At 15 days two types of lesion were seen, sometimes in adjacent sutures in the same slide (*Fig.* 536 A). In one there was polymorph digestion with active granulations; in the other condensed granulations and giant-cell attack. The pus had disappeared by 25 days, and only giant cells remained to attack the suture. By 35 days the area of cross-section of the suture was reduced by half (*Fig.* 537 A). No suture remained in many of the lesions at 50 days, but there was still a quarter of it left in some. A fragment of suture was seen in one section at 70 days and one at 90 days, but all the others at 70, 90, and 100 days showed merely a narrow scar.

5 days the gut was covered by a few polymorphs and some blue debris, but by 7 days there was a true pyogenic reaction causing superficial erosion. At 10 days the granulations were more condensed and the pus was scantier, but up to a quarter of the suture had been digested. By 13 days the granulations formed a narrow ring and there was only a little free pus (*Fig.* 535 B). At 15 days the pyogenic reaction had returned with increased violence. The ring of granulations was thicker and more active than ever. The lesion was now twice the diameter of the suture, which was bathed in fresh pus and reduced by about a third (*Fig.* 536 B). This unexpected result caused us to repeat the 15-day experiment and we again had the same picture which made it unlikely that the

return of the pyogenic reaction was due to infection. By 25 days the lesion was reduced in size to about the diameter of a suture. Only about a quarter of the gut was left and this was being digested in some places by fresh pus and attacked in others by giant cells. At 35 days pus was still forming around the suture (*Fig.* 537 B), but at 50 days attack was by giant cells only. Suture fragments were present within giant cells at 70 days, but at 90 and 100 days scar only remained.

Table I.—Tensile Strength of 0 Plain Catgut

Days since Implantation	Number of Tests	Average Tensile Strength (lb.)	Number of Tests where Tensile Strength was Nil
Dry unimplanted	20	9·42	0
1	20	5·34	0
2	19	6·00	0
3	20	3·12	0
5	19	4·30	0
7	20	1·52	3
10	17	1·75	3
15	20	0	20
20	20	0	20

Table II.—Tensile Strength of 2 Plain Catgut

Days since Implantation	Number of Tests	Average Tensile Strength (lb.)	Number of Tests where Tensile Strength was Nil
Dry unimplanted	20	17·83	0
1	20	9·44	0
2	20	10·56	0
3	19	5·79	0
5	20	5·24	0
7	20	3·53	1
10	20	1·82	3
15	20	0·52	8
20	16	0	16
25	16	0	16

Tensile Strength.—The tensile strength in pounds of the samples of catgut used at varying time intervals in the rat's muscle are given in *Tables I–IV*.

DISCUSSION

Size 2 plain catgut remained reliable for a little longer than the finer grades. The height of the inflammatory response was delayed until 4 days and most of the sutures were reasonably strong until 5 or 6 days. Implants of size 0 plain catgut produced the same histological appearances as 3/0 and 5/0 implants, but the tensiometer showed that size 0 remained strong for longer, and, like size 2, could be relied upon in most cases for 5 days. This does not necessarily mean that the larger sizes are more useful in surgical practice as it has been shown by Bates (1939) that they may cause greater delay in healing.

The early pyogenic reaction to plain catgut in the tissues followed by loss of its tensile strength has been observed by everyone who has implanted this material into animals. Bates (1939) wrote that plain catgut excited a prompt, violent, exudative foreign-body reaction which delayed the appearance of fibroblasts and so delayed healing. Large plain catgut was absorbed practically as fast as the smaller sizes and any size failed to hold throughout the time wound support was needed. Burke (1940) stated that plain catgut lost about 90 per cent of its strength within 6 days, and Madsen (1958) suggested that the use of plain catgut should be very limited as it was unreliable. If human and rat tissues are similar in their behaviour to this material, we strongly support this view.

Table III.—Tensile Strength of 0 Medium Chromic Catgut

Days since Implantation	Number of Tests	Average Tensile Strength (lb.)	Number of Tests where Tensile Strength was Nil
Dry unimplanted	20	10·53	0
1	20	7·17	0
3	20	5·71	0
7	20	5·56	0
10	20	7·12	0
15	20	3·71	0
20	18	3·78	0
30	15	1·63	3
40	20	0·21	14

Size 2 medium chromic catgut, unlike the finer grades, caused a pyogenic reaction in the tissues around it. Unlike the reaction to plain catgut, this was not immediate but delayed for 7 days. About a quarter of the gut was digested by pus and by 13 days the inflammation had subsided. At 15 days it returned with some violence, and fresh pus continued to digest the suture until 35 days. Size 0 medium chromic catgut behaved like the smaller sizes until 13 days, when some of the lesions showed pus and inflammation. In the inflamed lesions the gut was digested by pus; in the quiet lesions it was removed by giant cells. All the pus had been absorbed by 25 days.

Table IV.—Tensile Strength of 2 Medium Chromic Catgut

Days since Implantation	Number of Tests	Average Tensile Strength (lb.)	Number of Tests where Tensile Strength was Nil
Dry unimplanted	20	18·66	0
1	20	15·51	0
3	20	14·45	0
5	20	13·60	0
7	20	13·24	0
10	20	12·07	0
15	20	10·35	0
20	14	6·11	0
30	15	4·57	0
50	16	0	16

Every sample of chromic catgut used in this work was treated with chrome salts in the 'ribbon' stage before it was spun. Small sizes were made by spinning a few ribbons together, larger sizes by spinning several ribbons. It is unlikely, therefore, that the pyogenic reaction was caused by incomplete tanning. It is possible that the tissues were rendered hypersensitive by the large bulk of catgut; the delayed reaction and its inconstant appearance with size 0 gut make this hypothesis more likely. Pickrell and Clay (1944) were unable to induce hypersensitivity in rabbits or guinea-pigs by injecting into them a colloidal suspension of plain catgut. Sewell and Craver (1955), after repeatedly injecting guinea-pigs with a suspension of powdered catgut, implanted 3/0 plain catgut. They claimed that the implants produced a greater pyogenic reaction in

from 7 to 15 days than similar implants in uninjected control guinea-pigs. However, by 25 days a brisk pyogenic reaction had developed around the implants in the control animals, suggesting that they had become sensitized by the implant itself.

Harvey (1934) favoured the use of fine sutures in surgical practice and gave his reasons as follows: "... it is apparent that the approximation of tissues must be obtained without tension on the line of healing. If tension is unavoidable this must be taken up at points at a distance from the incision. The question here is what size and type of suture should be used in holding the tissues in approximation. Certainly it is stupid to use one that is stronger than the tissue through which it passes. Fascia will rarely sustain a suture pull of more than 5 lb. and this is likewise true of tendon, while 'C' twisted silk of a standard grade has a tensile strength of 6 lb. and 00 catgut an equivalent amount. ... If this is true of these dense tissues, it is much more so for muscle and fat, and one is not justified in using anything but the finest sutures in these structures. To employ, as is customarily done, heavy braided silk or No. 2 catgut in approximating wounds is as unintelligent as mooring a rowboat with a ship's cable."

We have shown that only the finer grades of chromic catgut can be implanted in rat tissues without causing any pyogenic reaction. We share with Bates (1939) the view that "... the perfect suture will hold adequately for those fourteen days and then disappear, but it must not during its stay in the tissues adversely affect the rate of wound healing. ... Wound support and healing are most satisfactory when the smallest chromic suture is employed."

SUMMARY

Coarse catgut was implanted into the lumbar muscle of rats, which were afterwards killed at intervals between 1 and 100 days. The tensile strength of the catgut was then measured and the material taken for histological examination.

The results were compared with those of identical experiments on fine catgut reported in a previous paper. Coarse plain catgut caused almost the same prompt pyogenic reaction as fine plain catgut and lost strength almost as rapidly. A delayed pyogenic reaction was produced by coarse medium chromic catgut which was absent in the implants of the finer grades. All the medium chromic gut used remained reasonably strong for at least 14 days.

It is concluded that the fine sizes of medium chromic catgut are the most satisfactory absorbable suture materials we have tested.

Acknowledgements.—Our thanks are due to the Directors of Ethicon Ltd. and to the Royal College of Surgeons of England for permission to publish this work; and to Professor G. J. Cunningham, M.B.E., Department of Pathology, Royal College of Surgeons of England, for his help and advice; and to Mr. A. L. E. Barron of the same department for the photomicrographs.

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WOUND SHAPE AND TISSUE TENSION IN HEALING

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"L'ulcère rond ne reçoit cure, s'il ne prend une autre figure."—Ambroise Paré.*

THE work of Carrel (1910) gave a great impetus to the study of wound healing and the contraction process that occurred. It is unfortunate that the mathematicians who then took up the problem did not heed the simple observations of the practical surgeon from several centuries earlier. It is only recently that wound shape has received adequate attention from Billingham and Russell (1956). Similarly the effect of wound tension has been ignored. Hippocrates in *The Coan Prognosis* remarked: "The most difficult fistulæ are those which occur in the cartilaginous areas where there is little fleshy substance. ... The easiest to treat are those which occur in the soft fleshy places and places free from tendon." (Chadwick and Mann, 1950.) Not until Langer (1861) and Loeb (1898, 1920) was this to be revived as a scientific observation. The former's observations on skin tension lines and the orientation of incisions have now with modification become surgical commonplaces (Kraissl, 1951). Loeb has yet to receive full credit for demonstrating the changed pattern of healing when rigidity of underlying tissues or other causes change the contraction process.

Wound healing consists of three quite distinct processes—epithelial regeneration, contraction, and the formation of granulation tissue, which leads to the final cicatrix. Various conditions determine which of these three proceeds most rapidly. Consequently, different types of wounds often appear superficially to heal in a different fashion as, for example, when they are of unequal sizes. Since the various processes

* "Round ulcers do not heal easily unless they are drawn into another shape."

vary in their time of onset, it is obviously possible for one apparently to complete healing before the others have begun, although they may take part in the process of remodelling of the existing tissues is equally or more important in effecting closure of the defect.

Epithelial repair begins very rapidly (Cameron, 1951) and an incised wound may be completely covered by epithelium within twenty-four hours.

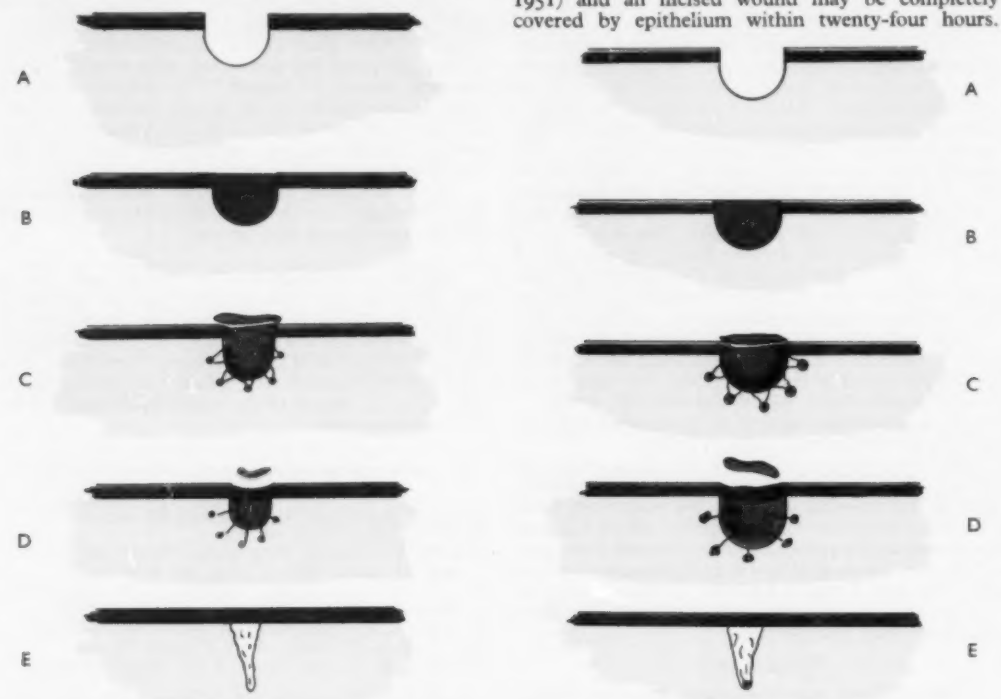

FIG. 538.—Diagram to show healing (*left*) with and (*right*) without contraction. The former is mainly a process of remodelling, the latter is one of regeneration of tissue. In both cases there is a secondary contraction when the cicatrix shrinks. A, Cavity. B, Cavity filled by clot. C, Invasion of clot by blood-vessels to form granulation tissue. D, Cavity closed by connective tissue and covered by epithelium (scab detached). E, Cicatrix.

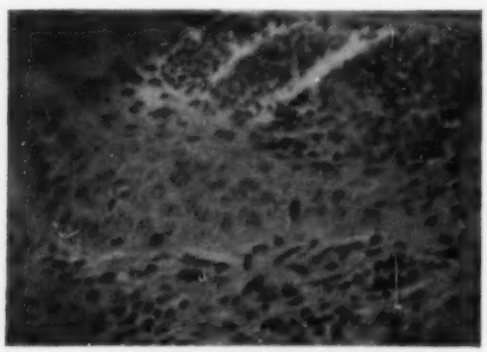

FIG. 539.—Section of 5-day wound in guinea-pig showing epithelium growing under scab.

unnoticed at a later stage. Not all need occur, however, and much confusion is caused by failure to realize this. *Fig.* 538 shows the difference between healing with and without contraction. In the latter, healing is a process solely of regeneration; in the former, This is a process of cell multiplication, the cells at the cut edge multiplying, spreading down to the base of the wound, and then extending across it beneath the scab. This can be seen easily in *Fig.* 539. In some lower animals, however, repair of epithelial defects can occur even before cell multiplication has taken place; the adjacent epithelium detaches itself from its underlying base, slides across and becomes re-attached, behaving as though it were a carpet held down by a number of snap fasteners (Weiss, 1956). Epithelial growth and healing have often been cited as the cause of the contraction process of the wound, but it is easy to show that this is not so (Watts, Grillo, and Gross, 1958) by daily removal of the scab; this tears away the regenerating epithelium without change in the rate of contraction. The spread of epithelium appears to be almost completely independent of the other processes going on in the wound. The rate of growth was studied by Henshaw and Mayer (1943), who found that in a wound which did not contract epithelial coverage occurred at a steady rate as it spread away from the wound edges. On this basis they evolved the principle of the 'greatest inscribed circle' (*Fig.* 540). The outline of a wound is traced and the largest circle found

that just touches the perimeter without crossing it. The time for full epithelialization is directly proportional to the radius of this circle, since this is the farthest distance which epithelium will need to grow before the wound area is completely covered.

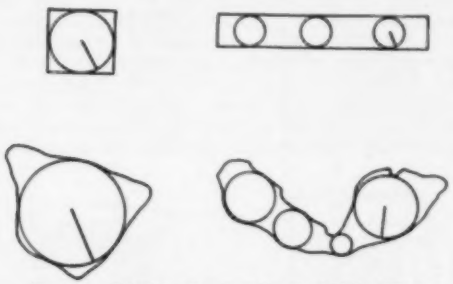

FIG. 540.—Principle of greatest inscribed circle. The two rectangular areas have the same area. The time taken for epithelialization, however, will not be the same. It is directly proportional to the radius of the largest circle that can be drawn within the wound. This is the greatest distance epithelium will have to grow. The irregular figures can be similarly assessed as shown. This theory assumes that epithelialization alone is acting to reduce the wound area.

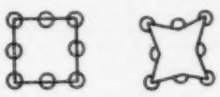

FIG. 541.—Change in shape of a square wound when contraction occurs. The circles represent tattooed markers made before the central skin is excised. Note the remodelling which also occurs.

The contraction process was studied systematically by Carrel (1910). Unlike many of his disciples, however, he realized that contraction and epithelial repair were not synonymous and differentiated between the two by the simple technique of using experimental animals with pigmented skin. Abercrombie, Flint, and James (1954) have more recently introduced the principle of tattooing to show the same point; this has great advantages when using smaller animals, as uniformity in the animal and site may be achieved. These workers noticed when studying square wounds that as contraction proceeded the wound became stellate (*Fig.* 541); this observation is compatible with the theory that simple mechanical forces within the wound could be the cause of the contraction process. D'Arcy Thompson (1942) has demonstrated that a cube of gelatin when dried assumes a similar shape on cross-section (*Fig.* 542). That contraction is not due to dehydration was shown by Grillo, Watts, and Gross (1958), since the water concentration of the tissues remained constant (as might be expected on elementary physiological grounds). We have also shown that the thickness of the granulation layer in a full-thickness skin wound in a guinea-pig rises to about 0·4 mm. and then remains constant. This was done by correlating the area, the total granulation tissue weight, and specific gravity at varying stages of healing. This also would not agree with the theory of contraction by dehydration. Abercrombie, Flint, and James (1956) showed that the contraction was not produced by the rise in collagen concentration of the granulation tissue, since it could still occur in scurvy where no collagen whatever was formed. This has recently been confirmed by Grillo and others (1959). Abercrombie and others (1956) concluded that the mechanism lay in the cells. This supports Lindquist's (1946) earlier experiments (*Fig.* 543). In these tape was fixed to the skin along the perimeter of a wound and the healing process allowed to continue; incisions were then made along the edges of the normal skin. It was found that the granulation tissue in the centre retracted away from the skin. Unfortunately, these experiments were all made on very small wounds where the edge is hard to define, especially when unmarked. Billingham and Medawar (1955) have suggested that there is an intussusceptive growth process outside the wound area. This would contribute to contraction and healing by producing a centripetal force. This is, of course, very hard to assess since all measurements have to be made on tissues after removal from the animal. Cuthbertson (1959) has shown that stretching of the animal skin takes place over a wide area, but that it is more pronounced close to the wound.

FIG. 542.—Change in shape of a gelatin cube on drying. The figure on the right shows the drying cube in cross-section. Compare with *Fig.* 541.

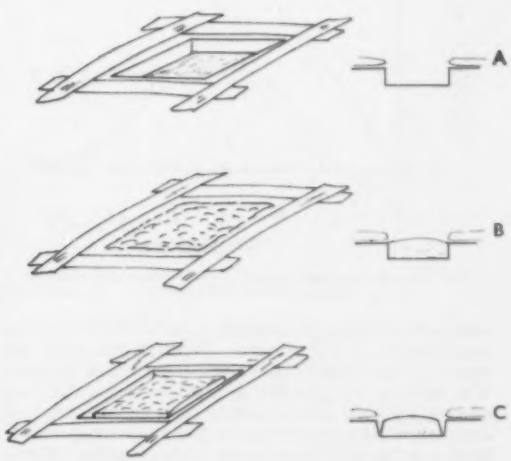

FIG. 543.—Lindquist's experiment on contraction. A wound is made as at A and tape fixed to the edge. This is then allowed to fill with granulations (B). An incision is then made along the edge of the skin, whereupon the granulations contract (C). The smaller diagrams show the various stages in cross-section.

It can be demonstrated by tattooing concentric squares around a wound before it contracts (*Fig.* 544). Watts and others (1958), in endeavouring to locate

the source of the contracting force, carried out a series of morphological experiments in which various parts of the wound were excised (*Fig.* 545). Whether the central granulation tissue was removed once or repeatedly the contraction process continued unaltered. An incision just outside the perimeter of the wound resulted in marked retraction of the skin around the wound to produce a new wound greater in size than the original. The forces in the surrounding skin are therefore not aiding contraction but pulling outwards, tending to produce distraction, not closure of the wound. Interference with the edge of the wound delays the contraction process. The moving force in fact appears to be located in a narrow zone or picture-frame around the edge of the wound; this moves across the underlying wound base, leaving little or no trace of its progress. Histological examination shows that it is an area of intense fibroblastic activity. Grillo, Gross, and Levine (1959) have recently shown that there is actual cell orientation occurring at this point.

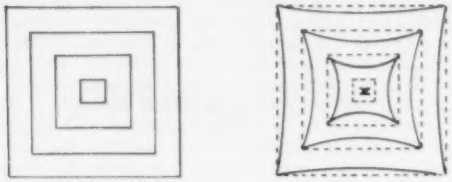

FIG. 544.—Diagram to illustrate the distortion when a wound contracts. The innermost square represents the wound, the outer ones lines tattooed around the wound. The second diagram shows the distortion after contraction.

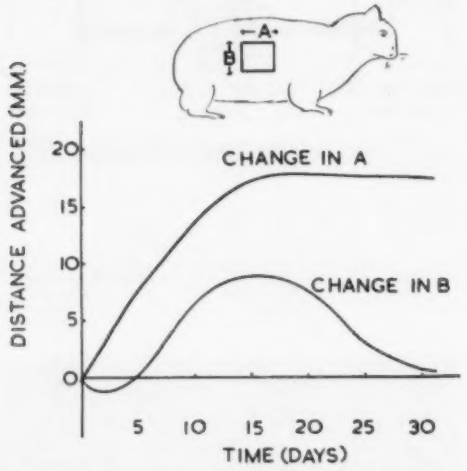

FIG. 546.—Linear measurement of edge advance in contracting wound in flank of guinea-pig.

Watts and co-workers noticed that a square wound in the flank of a guinea-pig did not actually close uniformly in all directions when contracting, but produced an elongated stellate scar at right angles to the long axis of the animal. Measurement of the rate of movement of the vertical edges of this wound showed that it was constant until the margins met (*Fig.* 546). Movement of the vertical edges, however, began before that of the horizontal ones so that the latter were at first pushed outwards. Their movement, when started, was uniform, but progress was stopped when the vertical edges met and the horizontal ones were then pushed outwards again to a final position, approximately the same as in the original wound. If a wound similar to this is made with its axis rotated 45°, all the sides move uniformly (*Fig.* 547), but movement is almost entirely along the longitudinal axis of the animal. It seemed to us that this behaviour was probably related to the distribution of Langer's lines of skin tension. These were therefore plotted for the guinea-pig (*Fig.* 548).

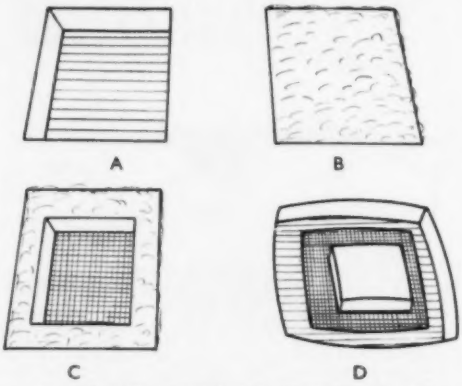

FIG. 545.—The morphological experiments of Watts, Grillo, and Gross (1958). A wound is made as at A down to the muscle layer. This is allowed to granulate as in B. If the central granulations are excised (C) no change occurs at the time and contraction proceeds as normal. If a narrow strip of skin and granulations is removed the wound becomes distracted (D).

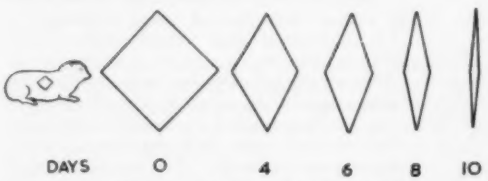

FIG. 547.—Diagram to show contraction in a 'tilted' square wound.

Bearing in mind the above observations we now compared the rate of contraction in wounds with different orientation. Incised wounds were made in the flank and sutured at once with interrupted stitches; on one side in the long axis of the animal, on the other side at right angles to this. The ends were marked by tattooed lines at right angles to the wound. Invariably it was found that the incisions across Langer's lines contracted by about 10 per cent of their length. Wounds at right angles to this, i.e., parallel to Langer's lines, did not contract at all. Wounds at intervening angles showed greater or

lesser degrees of contraction depending on the angle they made with Langer's lines. This experiment was then repeated, but using oblong incisions through the whole thickness of the skin instead of incised wounds. These measured 4 cm. × 1 cm. The results when these contracted are shown in *Figs.* 549, 550. It was interesting to see that the resultant scar was closely similar in shape whichever way the wound was alined.

It was also surprising to find that when the contracting edges were farther apart, i.e., when they were the short ends in a horizontally placed wound, the edges moved faster. Since we knew from previous experiments that excision of the central granulation tissue did not affect the rate of contraction, it is obvious that this must be caused by factors in the wound edge or outside it. The most likely cause of this is tension. Weiss (1933) has shown that several factors, such as ridges on a basement membrane across which cells are advancing, can affect orientation of cells. Since Grillo, Gross, and Levine (1959) have shown that such orientation does occur, tissue tension could obviously produce this change. The next experiment was to measure the effect of tension which prevented wound edges from advancing; this was achieved by sutures passed through the skin near to the edge of a square wound and fastened to the skin of the animal well ahead of and behind the wound. In placing these sutures some degree of distortion of the wound was produced so that it became a little elongated in the long axis and contracted in the vertical axis; these sutures maintained their pull for five to seven days before cutting out (*Fig.* 551). During this period the lines of skin

FIG. 548.—Langer's lines in the guinea-pig. These were plotted by using a round punch and by small stab wounds. These are true 'Langer's lines' as they were measured on the dead animal (*see* Kraissl, 1951).

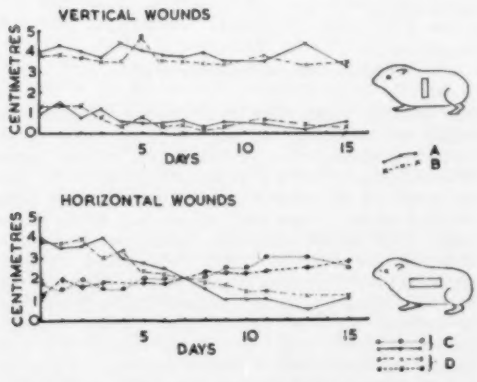

FIG. 549.—Contraction in elongated wounds with different axis orientation.

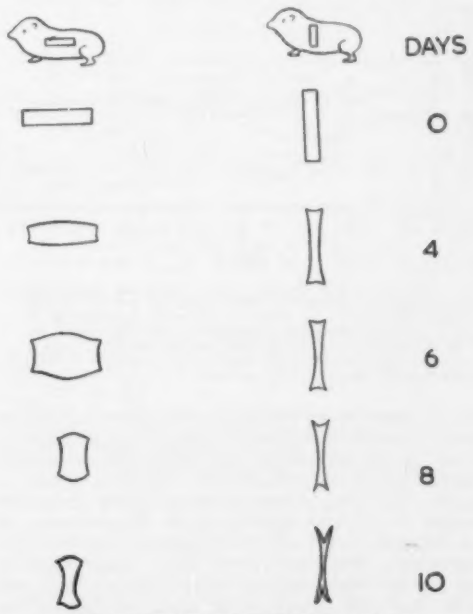

FIG. 550.—Graphic representation of *Fig.* 549.

tension are rotated through 90° by the pull of the stitches. While this is so the horizontal edges advance, unlike the normal situation when the vertical ones move (*Fig.* 552). After the stitches cut out (spontaneously), the vertical edges move again rapidly, and the final scar is the same as in controls where no tension is applied. It would thus appear that the factor determining the direction of contraction is a purely mechanical one from tension. Normally the tension is determined by the animal's bodily configuration.

From the experiments and observations described and from measurement of the total amount of granulation tissue in a wound when correlated with contraction (*Fig.* 553), it appears that granulation tissue is a transient structure, and that before wound healing can become complete some, if not most, of the granulation tissue must be removed. Thus, although granulation tissue provides a basis for the final scar and fills in defects not closed by the contraction process, it is obvious that the less granulation tissue that forms the more easily can the final healing occur, since less tissue must be formed and removed in the process. This is one of the fundamental principles underlying the suturing of wounds. If a large amount of granulation tissue survives then an indolent

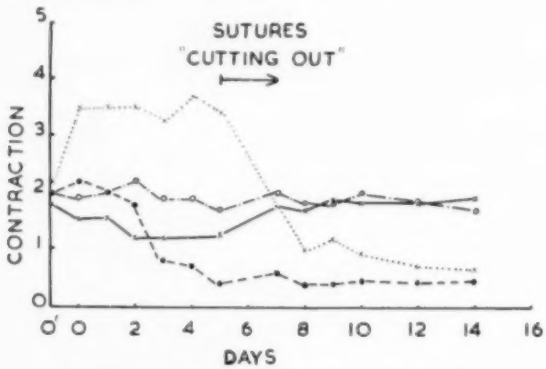

FIG. 551.—The effect of tension preventing contraction. The tension is applied by sutures after the wound has been made and explains the change in size of the wound at O' O. x . . . . x, Horizontal axis of experiment; x——x, Vertical axis of experiment; •— —•, Horizontal axis of control; ○— · —○, Vertical axis of control; O', Before tension applied; O, After tension applied.

wound may result. This is seen when factors are present which stimulate an excess of granulation tissue or prevent normal contraction occurring with the absorption of granulations. The first is seen in sepsis. The latter is seen by virtue of the site of the wound or tensions applied to it. Disturbance, as for example in lack of rest or support, also prevents contraction occurring. From these observations it can be seen that wounds along Langer's lines will show less granulation tissue since the contraction process tends to narrow the cavity. Those at right angles are not so compressed. Hence it is not surprising that the latter are less linear in their final state. Also, of course, the contraction in their length explains the bow-stringing effect sometimes seen when wounds cross a depression. The formation of keloids in wounds across Langer's lines is also explained.

It has been pointed out above that contraction is essentially a cellular process affecting the edge of the wound. Although this is accompanied by remodelling,

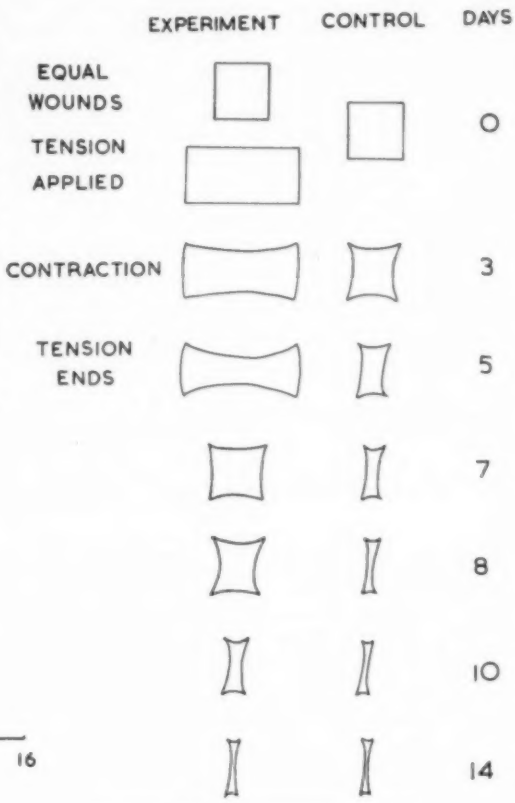

FIG. 552.—Graphic representation of *Fig.* 551.

the wound edge does not change as much as might be expected. If the perimeter of a square wound is measured it is found that as contraction proceeds there is not a great difference in the total perimeter of the wound, the reduction usually being at most about 25 per cent, often only about 10 per cent. The actual reduction occurs as a result of distortion of the edge rather than contraction; this explains why a circular wound does not heal as well as the irregularly shaped wound unless some force is present whereby the circular wound becomes converted to an oval one. If the wound is a large one in the flank, then of course distortion can occur, since some parts of the perimeter will not be able to migrate as quickly as others. When there is no overriding distorting force due to the natural tensions in the skin, however, the contraction can only occur by actual shrinkage of the perimeter. This is a slow process, especially so since the longer the healing takes, the thicker the plaque of dense collagenous

scar tissue which forms in the base of the wound and hinders the movement of the edge. Hence the aphorism of Paré. (Singer, 1924.)

The effect of the tension along Langer's lines on the closing of a wound can be likened to some extent to the repair of a hole in a bead screen. If the gap is closed from side to side there is no problem since it is merely necessary to draw the strands apart a little on either side of the closure. Repair at right angles

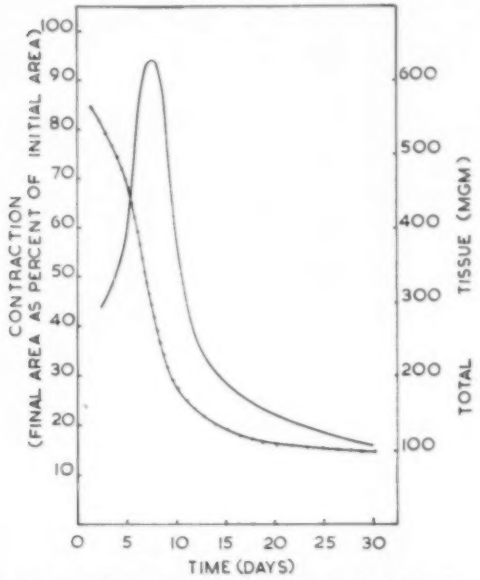

FIG. 553.—Contraction expressed as percentage of original area and the total amount of tissue in a wound.

to this, however, would pull the screen away from the floor or the ceiling by the drag on the rigid thread carrying the beads. The usual shape of a wound which is chosen by surgeons, an ellipse with its long axis along Langer's lines and with tapered ends, is therefore on mechanical grounds most rational. It is, moreover, the most suitable, for the physiological reason that the natural contraction process of healing tends to occur across its axis. The failure of trigonometric calculations in plastic surgery such as those advocated for Z-plasty (McGregor, 1957) is also explained.

Perhaps the most important observation on the process of contraction and regeneration is that if the wound edges cannot be brought together at the very beginning by sutures, then healing will probably never occur satisfactorily, since the tensions which prevent this closure will also be sufficient to prevent the pull of the contraction process. The gap remaining in these cases can therefore only be filled by a block of scar tissue which can be covered by a single layer of epithelium growing out from the wound edges. Such a wound is inherently defective, since it lacks the normal constituents of the skin almost completely.

SUMMARY

1. Wound healing consists of two processes: (1) Regeneration; (2) remodelling.

2. Granulation tissue is in great part a transient structure.

3. Wound contraction is related to cell orientation and is effected by movement of the wound edges.

4. The direction of contraction can be altered by superimposed tensions.

5. Contraction normally occurs at right angles to Langer's lines of skin tension.

6. Contraction is in a great measure produced by change in shape of wounds.

7. The planning of wounds and wound shapes in relation to Langer's lines is sound in fundamental principles based on wound contraction.

8. Keloid formation has a similar foundation in normal physiology.

9. When wound edges cannot be approximated *per primam* by sutures, healing is impaired.

I should like to thank Professor F. A. R. Stammers for his help in preparing this paper and for the way that he and Mr. d'Abreu made the work possible in the first place. I also owe much to Dr. E. D. Churchill, Dr. H. C. Grillo, and Dr. J. Gross, in whose generous and pleasant company the foundations of this paper were laid at the Massachusetts General Hospital. My thanks are also due to Miss Cole and Mr. Dee for help with the diagrams.

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CHANGES IN GASTRIC ACIDITY AND EPITHELIAL REGENERATION FOLLOWING EXCISION OF THE GASTRIC MUCOSA IN THE DOG

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THE removal of a large part of the stomach is still the most frequently used surgical method to reduce gastric secretion. The reduced capacity of the stomach after partial gastrectomy often leads to undesirable complications which might be avoided if the mechanical functions of the stomach could be left intact.

With this in mind we have attempted to reduce the acid-secreting power of the stomach by the removal of only the gastric mucosa, allowing regeneration of the gastric epithelium. It was hoped that the regenerated epithelium would not produce a return of gastric acidity to previous levels.

The present paper records the technique of this procedure and the results obtained. Although there was a great reduction in gastric acidity initially, this did not remain permanent. Histologically, the mucosa also showed a return to normal with differentiation of acid and pepsin-secreting cells in the regenerated mucosa.

MATERIALS AND METHODS

Fifteen adult mongrel dogs were used in these experiments. The dogs were obtained about two weeks before operation and were weighed daily. They were fed on a diet of raw and cooked meat and milk, the amount of food and fluid taken by each dog being measured at the end of each day.

Histamine secretory tests were carried out twice before operation and at roughly three-week intervals after the operation up to three months. After this time two animals were followed up to six months with tests every five weeks. Material for histological examination was obtained at the time of operation, and at the conclusion of each experiment when the animal was killed.

Histamine Tests.—The histamine secretory tests were carried out as follows: The dog was starved for 18 hours and was then anæsthetized by an intravenous injection of sodium pentobarbital (1 c.c. of 1 per cent sodium pentobarbital per 5 lb. bodyweight). This dose of pentobarbital lasted sufficiently long to cover the whole of the test. A double-lumen intragastric tube was then put in place. The position of the tube was checked by feeling the abdomen while moving the tube in and out, and by injecting air down the tube and feeling for abdominal fremitus (Milton, 1958). The outer tube was connected to a continuous suction apparatus and air was allowed to enter the stomach by the inner tube. Half an hour after the insertion of the tube the collected sample was removed and discarded. If the tube was blocked by mucus it was washed through with water.

The animal was next given a subcutaneous injection of histamine acid phosphate, 0·5 mg. kg. bodyweight. (These large doses of histamine were found to be necessary to produce maximal effect in the presence of pentobarbital.) Samples of the gastric aspirate were taken every 15 min. for the ensuing 2 hr. The volume of the samples was measured and the *p*H of each specimen was determined by a glass electrode *p*H meter. The samples were then titrated against *N*/10 NaOH, using Topfer's reagent and phenolphthalein as indicators.

The Surgical Procedure.—The dogs were anæsthetized with sodium pentobarbital and an endotracheal tube was put in place. A midline incision was used to expose the stomach. The vessels on both curvatures were ligated and cleared from the stomach wall over a distance of about 3 cm. at the level of the incisura and the stomach was completely divided across between these areas. The pyloric antrum was then packed and covered with swabs. The body of the stomach was turned almost inside out, and the mucosa was dissected free from the muscularis mucosæ. Approximately seven-eighths of the stomach was denuded of epithelium, only about 3 cm. at the cardiac and pyloric ends being left intact. The ease with which the mucosa could be stripped varied slightly from animal to animal. The procedure took, usually, about an hour. At the conclusion of the dissection biopsy specimens were taken from each side of the stomach wall and from the excised mucosa and fixed in 10 per cent formol saline. The stomach wall was sutured with chromic catgut in two layers. The abdominal incision was closed with silk and at the conclusion of the operation each animal was given an intramuscular injection of penicillin (500,000 units) and streptomycin (0·5 g.). The blood-loss was measured by weighing the swabs before and after the operation. Where the blood-loss exceeded about 300 ml., the dogs were given 500 ml. of 5 per cent glucose in 0·85 per cent saline and 2 ml. Ferrivenin intravenously.

Histological Examination.—When the animals were killed the stomach was removed, washed through, and distended with 10 per cent formol saline. After fixation the stomach was opened along the greater curvature, and the suture line as well as the mucosa was examined with the naked eye and with a watchmaker's lens. Six pieces of stomach wall were taken for histological examination, one from the residual normal mucosa at each end and four from the area which had been denuded of mucosa.

The histological material was paraffin-embedded, cut, and stained with hæmatoxylin and eosin. In some instances Motteram's pepsin stain (Motteram, 1951) was used.

RESULTS

The majority of dogs stood the operation well, and none of them showed any evidence of post-operative oozing of blood from the stomach. The mean blood-loss during the operation was 280 ml., the maximum being 700 ml. The animals showed a considerable decrease (approximately 25 per cent) in weight during the first three weeks after operation,

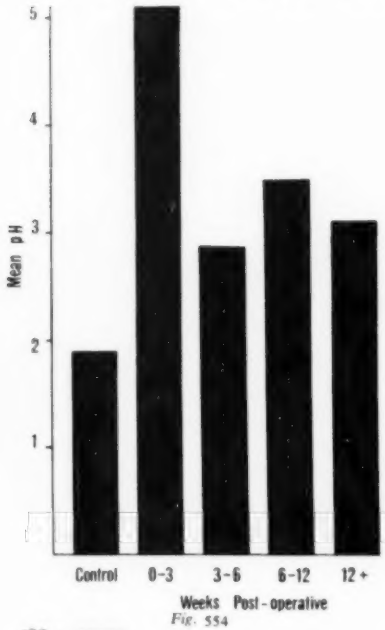
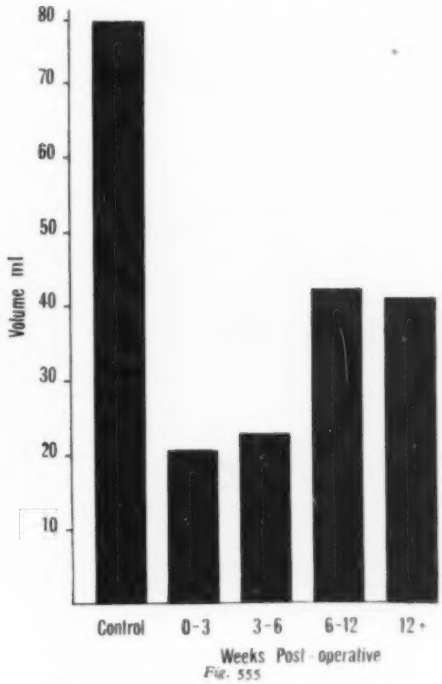


Fig. 554



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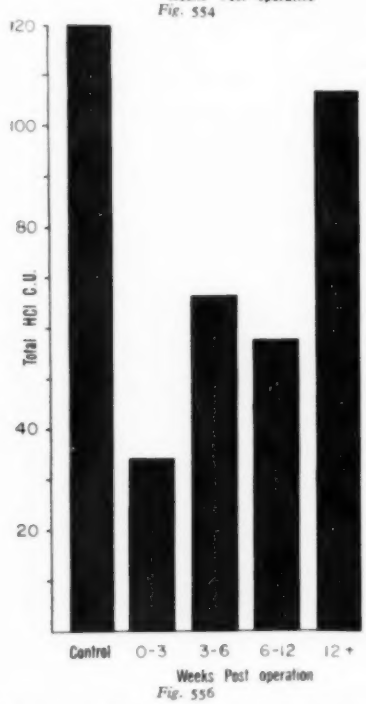

Fig. 556

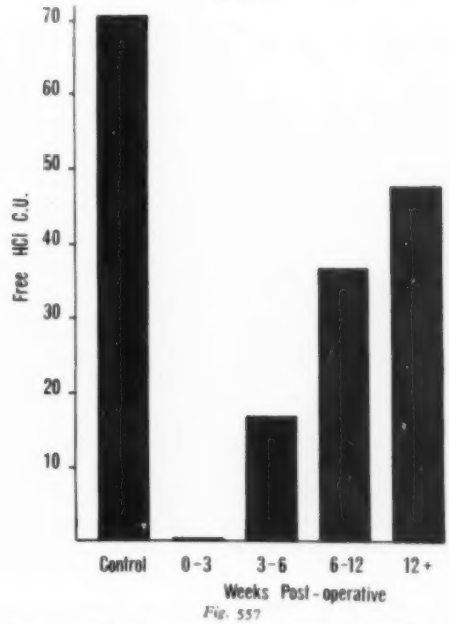

Fig. 557

FIGS. 554–557.—The serial changes in mean *p*H (*Fig.* 554), volume of gastric secretion (*Fig.* 556), total acid output (in clinical units) (*Fig.* 555), and free acid output (in clinical units) (*Fig.* 557).

At three weeks the mean *p*H had risen to 5·1, the volume of secretion had fallen to 20·7 ml., the total acid secretion had fallen to 34·5 clinical units, and the free acid to less than 1 clinical unit.

Later, there was a progressive return towards normal. The mean *p*H fell and the volume and acid output rose steadily. The relatively high reading of mean *p*H at 12 weeks in *Fig.* 554 is excessively influenced by an unusually high reading obtained in one animal.

and even after three months the remaining animals were still 10 per cent below their pre-operative weight. *Table I* shows the considerable decrease in the food and fluid intake during the first few weeks in two typical dogs.

Histamine Tests.—*Figs.* 554–557 show the results of the histamine secretory tests. The pre-operative histamine tests showed a consistent pattern of acid secretion. The mean *p*H of the resting gastric juice was 4·0, and following the administration of histamine the *p*H fell to 1·5. The mean *p*H of all samples following histamine was 1·8 (*Fig.* 554). A mean volume of 80 ml. of gastric juice was produced in the two hours after histamine (*Fig.* 555). A mean of 120 clinical units of total acid was secreted (*Fig.* 556) and the free acid amounted to 72 clinical units (*Fig.* 557).

During the first three weeks after operation the response to an injection of histamine was much reduced. The mean *p*H was now 5·1, the volume of secretion 20·7 ml., 34·5 clinical units of total acid were produced, and the free acid was less than 1 clinical unit.

Subsequent histamine tests showed a slow return towards normal. Although the mean *p*H at twelve weeks and more, as shown in *Fig.* 554, was 3·1, this comparatively high figure was due to one animal which took longer to return to near normality. If this animal is excluded, the mean *p*H was 2·0. However, at twelve weeks in all animals both the volume and the total hydrochloric acid output were still below the pre-operative levels, the volume being 41 ml., the total acidity 106 clinical units, and the free acidity 47 clinical units.

Structural Changes in the Stomach.—

Macroscopical.—Following the removal of the gastric mucosa the raw area was smooth and had small amounts of blood oozing from tiny bleeding points. In those dogs which were killed within a week of the operation the denuded area was red, slightly granular, and covered with a patchy layer of fibrinous exudate. The outer margin of the defect was now seen as a thin white line with a smooth glistening surface behind, suggesting the ingrowth of epithelium. In the early stages it was difficult to see discrete islands of epithelium in the raw area. After 3 to 5 weeks the regenerated mucosa at the periphery appeared much thicker, and finely cystic or villous to the naked eye. At this stage the defect was mainly covered with a smooth pale lining suggesting regenerated epithelium, but there were distinct 'islands' of thicker, cystic mucosa here and there. Two months after the operation the raw area had been completely covered with quite thick mucosa and there were few signs that the epithelium had once been removed from most of the stomach. A few patches of cystic mucosa remained, but elsewhere the mucosa was indistinguishable from normal. The stomach appeared to be entirely normal in the

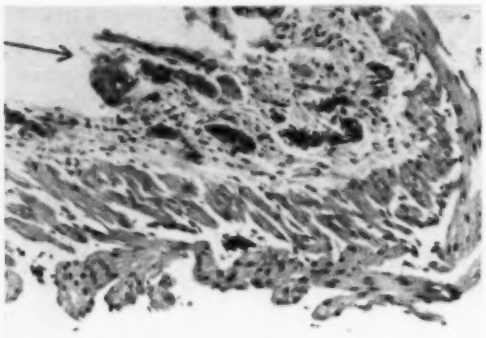

FIG. 558.—Section of portion of the stomach wall obtained immediately after stripping of the mucosa, showing the muscularis mucosæ and the residual overlying mucosa. In most areas only a fine layer of the lamina propria of the mucosa remained, but in this field a row of secretory cells from the tips of the glands (shown by the arrow) are also present. H. and E. (× 100.)

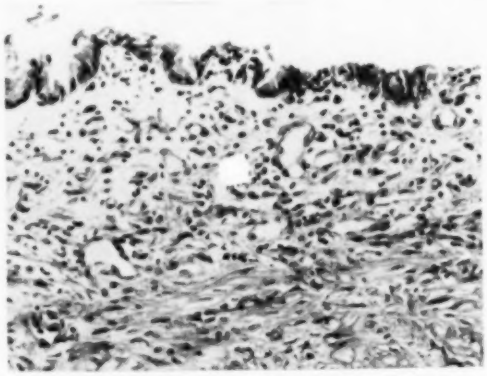

FIG. 559.—Portion of the denuded area of stomach obtained 5 days after mucosal stripping. There is a vascular granulation tissue over the muscularis mucosæ, largely covered by a single layer of cuboidal and columnar epithelium. H. and E. (× 100.)

Table I.—QUANTITY OF FOOD AND FLUID CONSUMED

(Values expressed are the mean daily consumption for one week)

DOG NO.	PRE-OPERATIVE	POST-OPERATIVE			
		1st Week	2nd Week	3rd and 4th Weeks	7th Week
11	Solid (g.) 6637	1540	3280	4252	5069
	Liquid (ml.) 154	41	59	120	140
19	Solid (g.) 4252	803	1784	2992	4131
	Liquid (ml.) 200	36	314	400	214

two animals which were killed six months after the operation, except for the line of the anastomosis, which appeared as a thin white scar.

In no animal was there any defect resembling a chronic peptic ulcer, as seen in man.

Microscopical Appearances. — Sections of the stripped mucosa taken at operation showed that the muscularis mucosæ had not been removed. Sections of the underlying stomach wall revealed a fine layer of connective tissue over the muscularis mucosæ, and rarely a few scattered acid and pepsin cells derived from the tips of the glands (*Fig.* 558). In sections of stomach obtained at different times after operation the following appearances were seen:—

FIG. 560.—Regenerated surface epithelium 2 weeks after mucosal stripping. The columnar epithelium is now folded and invaginated and forming tubular glands. H. and E. (× 100.)

At 5 days there was granulation tissue over the denuded area, mainly covered with a fibrinous exudate, but with a thin covering of at first flattened, and later columnar, epithelium in a few parts (*Fig.* 559). By 1½ to 2 weeks much of the surface was covered with a regular layer of epithelium. In some areas this was folded and invaginated, forming glands, and the thicker areas seen macroscopically at the outer edge and in 'islands' comprised quite large glands composed of clear columnar cells (*Fig.* 560). By 4–5 weeks most of the regenerated mucosa was glandular. In parts this was cystic in its deeper areas, or villous on the surface, and in others the deeper portions comprised quite tortuous glands resembling those of the normal prepylorus. In others, again, there were now cells resembling serous chief cells and parietal cells in the deeper portions. At 9–12 weeks most of the mucosa was of normal thickness and many of the glands contained chief and parietal cells (*Fig.* 561). At 22–24 weeks, except for a few areas overlying the anastomosis, the mucosa comprised regular, well-differentiated glands of chief and parietal cells, indistinguishable from normal (*Fig.* 562).

DISCUSSION

Several methods other than gastrectomy or vagotomy have been tried in man and animals to reduce the secretory capacity of the stomach, but all have produced only a temporary reduction. For example, external radiotherapy was used by a number of workers (Holman and Lewis, 1941; Ricketts, Palmer, Kirsner, and Hamann, 1948) and McGeorge (1950) tried the introduction of radium into the stomach. However, Joske, Finckh, and Wood (1955) found that the effects of radiotherapy lasted only 3 to 6 months, and Douglas, Ghent, and Rowlands (1951) considered that the hypochlorhydria produced in

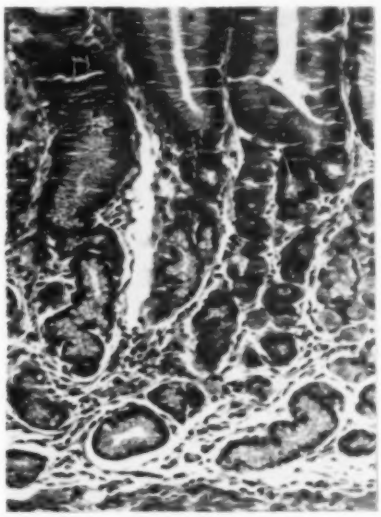

FIG. 561.—Regenerated mucosa obtained 9 weeks after operation. The mucosa is now of almost normal thickness and comprises well-formed glands composed partly of tall columnar cells and partly of well-differentiated serous chief and parietal cells. H. and E. (× 100.)

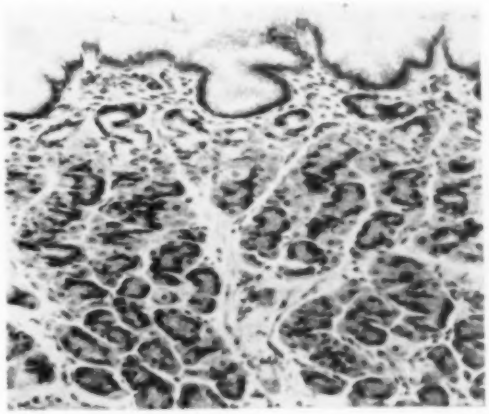

FIG. 562.—Regenerated mucosa obtained 22 weeks after operation. The mucosa contains normal glands made up of serous chief and parietal cells, and is indistinguishable from mucosa obtained before stripping. H. and E. (× 100.)

dogs by intragastric irradiation with P32 was probably not permanent.

On the other hand, Wynn Williams (1953) produced defects in the gastric mucosa of small laboratory animals by heat and observed the regeneration of the epithelium. He recorded a rapid replacement of the mucosa, but did not observe differentiation into acid- and pepsin-secreting cells in periods up to 60 days. Gunter (1950) reported similar findings in cats after producing mucosal defects with a flexible biopsy tube; there was regeneration of epithelium which appeared to be non-acid secreting. These findings suggested to us that the complete loss of secreting epithelium might not be followed by a return of normal secretory mucosa and the experiments reported here were designed to test this possibility.

Our results show that the gastric mucosa in the dog has remarkable powers of regeneration. Not only is the area stripped of mucosa rapidly covered with epithelium, but, given time, this proceeds to differentiate into the typical glands composed of acid- and pepsin-secreting cells which are specific for the lining of the stomach. This process of differentiation is accompanied by a return of acid secretion, and in three months the maximum acidity in the stomach is almost back to normal. In six months, while not yet normal in amount, the total acid secretion is considerable and is still increasing.

Our observations confirm the findings of Ferguson (1928), who showed that in dogs, though not in rabbits, stripped mucosa regenerated quite rapidly, differentiated, and eventually recovered its normal appearance. We, too, found that the raw surface at first became covered with a sheet of thin and then thicker cells; that these became invaginated, forming irregular, often cystic glands, and that after 4 to 5 weeks these glands became more regular and their cells differentiated into acid- and pepsin-secreting forms. We consider that these findings may also be applicable to man, since Caylor (1926) found similar flattened and cystic epithelium in the early stages of healing in chronic gastric ulcers.

The source of the new epithelium appears to us to be twofold. There is an advancing margin of cells which closes in the defect from its edges, and this is supplemented by islands of cells derived from the few deep glandular cells which were accidentally left during the stripping. It is generally supposed (Bizzozero, 1893; Brecher, Cronkite, Conand, and Smith, 1958; Payling-Wright, 1958) that the cells both of the surface of the mucosa and of the secretory parts of the glands are normally replaced by mitosis of the cells of the foveolæ, the newly formed cells then passing upwards and downwards to their new sites. However, it has also been shown by Ferguson that the differentiated cells of the margins of a mucosal defect frequently lose their specific features and rapidly return to a more primitive type. These cells in turn may undergo mitosis and add to the replacement epithelium, only to redifferentiate once more at a later stage. We consider that this explanation accounts for the islands of regenerated mucosa we observed in the centre of the denuded area in our experiments, for we are sure that only a few cells, of differentiated type and derived from the tips of the glands, were left to provide the central foci of new epithelium.

The present experiments show that the mere stripping of the gastric mucosa above the muscularis mucosa is not followed by permanent mucosal change, with reduced secretion, in the dog. Something more is required to produce chronic peptic ulceration or the appearances of atrophic gastritis seen in humans.

SUMMARY

Large areas of gastric mucosa were stripped by open operation in dogs in the plane internal to the muscularis mucosa, and the serial changes in gastric acidity and the regeneration of the mucosa were observed.

The operation was followed by a considerable fall in gastric acidity, but this returned progressively and in six months was little short of normal. The defect in the gastric mucosa was found to become rapidly covered with epithelium, which later developed into a glandular mucosa and which later still differentiated into acid- and pepsin-secreting cells. The sources of the regenerating epithelium were the edges of the defect and scattered glandular cells in its centre.

In no instance was the return of acid production or of the normal appearances of the mucosa unduly delayed and there was no residual peptic ulceration.

We are grateful to Professor John Loewenthal, Professor F. R. Magarey, and to Dr. H. O. Lancaster for their help in the preparation of this paper.

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HÆMOSTATIC PATCH IN PARTIAL NEPHRECTOMY IN DOGS

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PARTIAL nephrectomy is not infrequently the operation of choice in cases of localized renal pathology despite the fact that post-operative hæmorrhage and urinary fistula are well-recognized complications. A review of 155 partial nephrectomies led Hanley (1950) to state that "hæmorrhage is the commonest complication" and that "the fear of post-operative bleeding is the chief factor which prevents the operation from being employed more extensively": in Hanley's series there were 12 instances of clot retention in the bladder, 2 secondary nephrectomies for continued bleeding, and 9 cases developed temporary urinary fistula.

Hæmostasis is generally secured by interposing a plug of fat (Lowsley and Bishop, 1933), or free muscle, between the cut surfaces of the resected kidney and anchoring it in position by resuture of the carefully stripped and preserved renal capsule. Deep parenchymal sutures have also been advocated to approximate the cut surfaces of the kidney (Stewart, 1952), but some degree of renal parenchymal damage is unavoidable when such sutures are used.

To determine the efficacy of polyvinyl alcohol sponge as a hæmostatic patch 12 dogs were submitted to partial nephrectomy: the incidence of post-operative hæmorrhage and other complications was observed and the reaction of the host tissue to the foreign body was studied.

METHODS

A proprietary brand of polyvinyl alcohol sponge (Ivalon) was used. Six sheets, 6 cm. square and 0·2 cm. thick, were placed on top of each other between the disassembled halves of a 7 cm. × 6·5 cm. metal needle box. The box was wrapped under moderate compression with a gauze bandage and boiled for 40 minutes. This sterilized the sponge and compressed the 'Ivalon' sheets to a block from which hæmostatic patches of varying size and shape were easily fashioned.

The dogs weighed between 10 and 15 kilos, and were kept in routine animal quarters: prophylactic antibiotics (penicillin 300,000 units and streptomycin 250 mg. daily) were administered for five days following operation.

Anæsthesia was produced by intravenous sodium pentobarbitone with xylocaine infiltration of the loin. After extraperitoneal exposure of the kidney, the renal pedicle was clamped, the renal capsule reflected, and one or other pole was resected: a flat line of section was used in 8 cases while a wedge-shaped resection was performed in 4 cases. The caliceal system was sutured with interrupted 0000 braided nylon and the suitably shaped Ivalon patch applied. In 6 dogs (Nos. 7–12) the interrupted nylon sutures were left long, threaded through the patch, and tied over the sponge. The reflected capsule was then replaced and a continuous, over-and-over, 0000 braided nylon suture secured the circumference of the Ivalon sponge to the capsule and margin of kidney. While the sponge was held by the interrupted nylon stay sutures, the clamp on the renal pedicle was momentarily released to allow blood to impregnate the sponge, and this manœuvre was repeated after the circumferential suture was completed. After 5 minutes had elapsed the clamp was released again and there was a transient ooze of blood from the sponge: if bleeding was brisk the pedicle was clamped for a further 5-minute period and this invariably resulted in hæmostasis. The kidney could then be freely handled without bleeding.

The total duration of vascular occlusion never exceeded 40 minutes: no attempt was made to ligate or coagulate bleeding vessels in the cut surface and the wound was not drained.

The systolic blood-pressures recorded via the femoral artery ranged from 90 to 140 mm. Hg throughout the procedures.

The animals were sacrificed at varying intervals and the gross features of the resected kidneys were observed. The kidneys were then removed and sectioned through the patch, the naked-eye appearances of the interface between the kidney and the

Table I.—RESULTS

Dog Number	Period of Study (wk.)	Kidney			Post-operative Hæmorrhage	Complications	Distortion of Patch
		Side	Pole	Resection			
1	12	Left	Lower	Flat	Nil	Nil	—
2	53	Left	Lower	Flat	Nil	Nil	—
3	8	Right	Lower	Flat	Nil	Nil	—
4	48	Left	Upper	Flat	Nil	Nil	—
5	44	Left	Lower	Flat	Nil	Nil	—
6	Anæs. death	Right	Upper	Flat	Nil	Nil	—
7	42	Right	Lower	Wedge	Nil	Nil	—
8	40	Right	Lower	Wedge	Nil	Nil	—
9	41	Left	Upper	Wedge	Nil	Nil	—
10	12	Left	Lower	Wedge	Nil	Nil	—
11	16	Right	Upper	Flat	Nil	Fistula with pyonephrosis Nil	—
12	Anæs. death	Left	Lower	Flat	Nil	Nil	—

The systolic blood-pressures recorded via the femoral artery ranged from 90 to 140 mm. Hg throughout the procedures.

The animals were sacrificed at varying intervals and the gross features of the resected kidneys were observed. The kidneys were then removed and sectioned through the patch, the naked-eye appearances of the interface between the kidney and the

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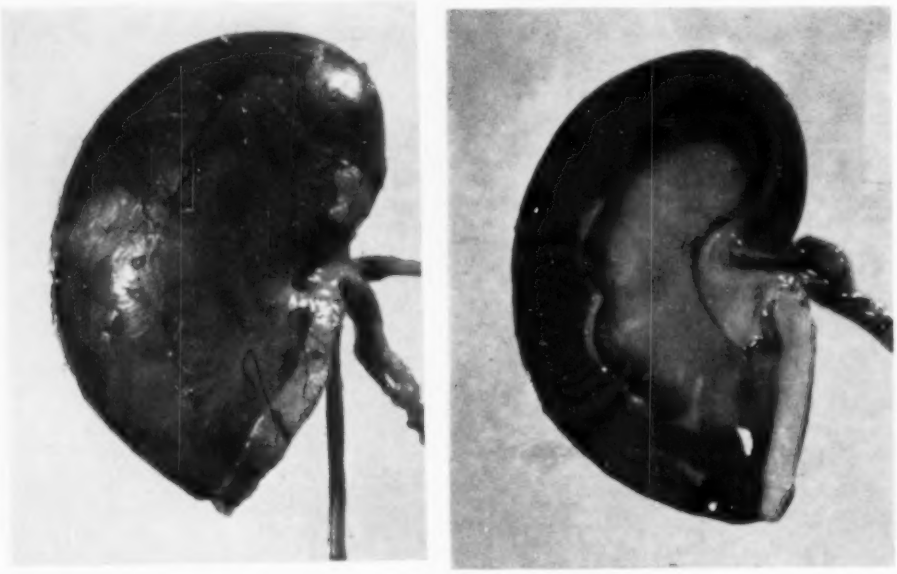
patch being noted. Because of difficulty in preparing histological sections of the Ivalon patch, study of the reaction provoked by the Ivalon was restricted to four specimens.

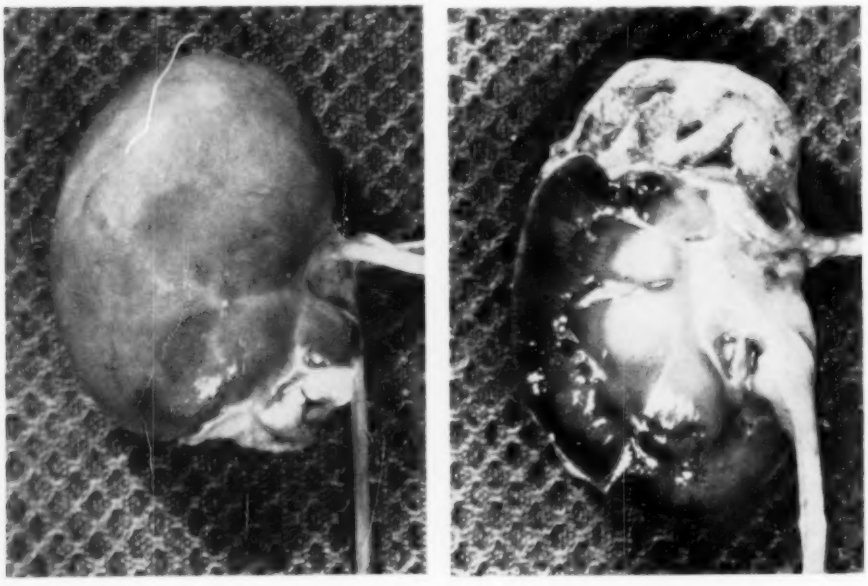
RESULTS (*Table I*)

1. Hæmostasis.—In all cases there was permanent hæmostasis. Two dogs (Nos. 6 and 12) failed to recover from anæsthesia which had been prolonged



A B

FIG. 563.—A, A flat Ivalon patch from Dog No. 5. B The patch seen on longitudinal section of the kidney.



A B

FIG. 564.—Two examples of distortion and corrugation of flat patches. A, Specimen from Dog No. 2. B, Longitudinal section of kidney (Dog No. 11).

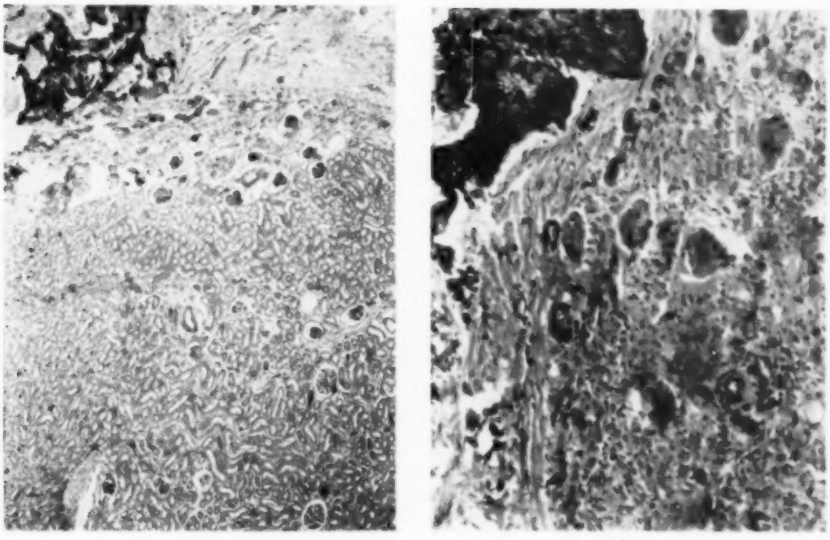
respectively for pyelographic studies and aortography. The specimens from these two animals were thus available for examination within 24 hours of operation: both showed no evidence of perirenal hæmatoma, but in one case (No. 6) the patch was ballooned by an underlying hæmatoma.

2. Fistula.—A fistula developed in 1 case (No. 10). This appeared nine weeks after operation and the animal was sacrificed after a further three weeks. The fistula was found to communicate with a pyonephrosis which had totally destroyed the kidney. This was the sole complication in the series.

3. Gross Examination of the Ivalon Patches.—In all cases the patches were covered with a thin fibrous capsule and were strongly adherent to the

FIG. 565.—A wedge patch on longitudinal section of the kidney (Dog No. 7).

FIG. 566.—Section of a wedge patch (Dog No. 8) to show the fibres enclosing and penetrating the synthetic sponge. V.G. (× 3.)



A B

FIG. 567.—A, Photomicrograph of specimen (Dog No. 2) showing layer of shrunken parenchyma interposed between sponge and normal kidney. H. and E. (× 30.) B, Higher magnification of shrunken layer infiltrated with lymphocytes and plasma cells. H. and E. (× 131.)

kidney. The sponge was firm and, compared with fresh Ivalon, difficult to section with a knife.

Flat Section.—In 4 instances the flat patches of Ivalon (Nos. 3, 5, 6, and 12) were smooth and retained their original configuration (*Fig.* 563): in 4 others the patch appeared distorted and corrugated (*Fig.* 564).

Wedge Section.—There was no distortion of the wedge patches (*Fig.* 565), but in 1 case (No. 10) in which a pyonephrosis had developed the inferior margin of the patch abutted on the ureteropelvic junction and was associated with a significant fibrosis and ureteric obliteration.

4. Histology.—Only 4 specimens (Nos. 1, 2, 4, and 8) were available for examination; the histological picture was the same in each of these. The patch was enclosed by a dense layer of collagen fibres which had permeated into the spaces of the synthetic sponge and had become continuous with the renal capsule (*Fig.* 566). There was no inflammatory reaction in the collagen or sponge, but interposed between the collagen and normal kidney tissue was a layer of shrunken renal parenchyma. This layer was 1–2 mm. thick and was infiltrated with lymphocytes and plasma cells (*Fig.* 567).

DISCUSSION

With this technique deep mattress sutures in the renal parenchyma are avoided and damage is limited to 1–2 mm. of adjacent renal tissue: such damage may be the result of ischæmia or of interference with drainage of the nephrons as a result of severance of their tubules.

Hæmatoma formation between the Ivalon and the cut surface of the kidney is liable to occur, but stay sutures, used to secure the centre of the patch to the resutured caliceal system, would appear to prevent this complication.

Four of the flat patches were corrugated and this was thought to result from changes in the shape of the cut surfaces of these kidneys. The more rigid wedge patches did not distort, a possible disadvantage, as pressure by the patch on the ureteropelvic junction might have contributed to the pyonephrosis in No. 10.

The physical structure of the sponge encourages the deposition of thrombus which fills its interstices and subsequently becomes organized to fibrous tissue. Microscopical examination failed to show evidence of rejection of the Ivalon by the host tissue.

This short series of experiments suggests that an Ivalon patch may be used for hæmostasis in the kidney when partial nephrectomy is carried out and confirms the observations of Jones, Nyhus, and Harkins (1958).

SUMMARY

In partial nephrectomy in 12 dogs, permanent hæmostasis was obtained using a patch of polyvinyl alcohol sponge. Histological appearances up to a year after operation showed minimal parenchymal damage and no evidence of rejection of the sponge by the host tissue.

Acknowledgements.—I wish to express my gratitude to Professor W. A. Mackey for his help and encouragement in this investigation. I am indebted to Professor W. L. Weipers and the staff of the Glasgow University Veterinary Hospital for assistance with the care of the animals; to Professor T. Symington for the assistance afforded in his department; to Mr. Towler for the photographs and Mr. Brackenbury for the photomicrographs. I also wish to thank Mr. R. Shackman and Dr. I. Doniach for their encouragement and advice in the preparation of this paper.

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SHORT NOTES OF RARE OR OBSCURE CASES

TRAUMATIC CHYLOTHORAX

REPORT OF A CASE

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TRAUMATIC chylothorax has always been viewed with fear and concern. Prior to Lampson's report (1948) of successful ligature of the thoracic duct, the condition had been stated to have a mortality-rate of 50 per cent if successfully treated, and 100 per cent if the surgical treatment had been unsuccessful (Hodge and Bridges, 1948). Harvey Cushing stated that the surgery of the thoracic duct was an impossibility (quoted by Seaman, 1954).

The condition is not common, and in the literature only one or two cases are usually reported in a single paper. Shumacker and Moore (1951) reviewed the literature and found only 70 cases of traumatic chylothorax. Seaman (1954), in a search for the frequency, found 110 cases reported up to 1949 and added 1 case of his own. Stranahan, Alley, Kausel, and Reeve (1956) described 3 cases of chylothorax, and 1 of these was due to damage of the thoracic

duct during mediastinal dissection for a case of thyroid carcinoma. A great number of causes have been attributed to the production of this condition, and among them are surgical trauma and fracture of the thoracic spine and left clavicle. Surgical trauma is probably the most common cause, especially the Smithwick operation for hypertension. Lee McGregor (1953), in a discussion on injuries to the large lymph-ducts, described 2 cases of damage to the thoracic duct occurring during the Smithwick operation. The injuries were discovered at the time of operation. The ducts were ligatured and each patient made an uneventful recovery. When one considers the large number of surgical procedures carried out in the vicinity of the thoracic duct, the incidence of damage to this important structure is extremely rare. The case reported is the only one seen in Kowloon Hospital for the past ten years. There is an average yearly admission of 10,000, of which 50 per cent are traumatic in nature.

CASE REPORT

C. M. M. (Kowloon Hospital No. 9610/58), a Chinese female, aged 25 years, was admitted on Dec. 4, 1958, because of self-inflicted stab wounds of the neck and abdomen in an attempt at suicide.

On Examination.—A well-nourished, well-developed Chinese female who appeared pale and was agitated and restless. Her mind was clear and she could answer questions well. The pulse was full and bounding. The rate was 110 per minute. Her blood-pressure was 130/100 mm. Hg and her respiratory rate was 24 per min., while her temperature was 96 F.

On her neck there were six incised wounds measuring 6–8 mm. in length and they appeared to be superficial. They were gaping, but there was no active bleeding. They were situated over the left supraclavicular region. The chest was hyper-resonant and the breath-sounds were diminished on both sides. Her heart was normal. A wound measuring 2·5 cm. in length was also found over the epigastrium and slightly to the left of the xiphisternum. There was marked tenderness and spasm over the upper part of the abdomen. There was no shifting dullness. The liver dullness was obliterated.

Portable X-ray examination of the chest revealed bilateral partial collapse of the lungs, with no demonstrable evidence of fluid (*Fig.* 568). Thoracentesis done in the ward produced about 600 c.c. of air from each pleural cavity.

At Operation.—Under general anæsthesia, with relaxant, the abdomen was opened by a midline supra-umbilical incision. As soon as the abdomen was opened the systolic blood-pressure dropped to 40 mm. Hg. An intra-abdominal source of hæmorrhage was looked for but there was no sign of injury to any of the abdominal organ. The stab wound in the epigastrium was limited to the muscles and a small tear of the peritoneum. The diaphragm was examined and it was found that air had accumulated in both pleural cavities in the interval between the thoracentesis and operation, and was probably the cause of the sudden drop in blood-pressure.

The diaphragm was opened on the right side after the right lobe of the liver had been retracted downwards by an assistant. The lung was found to be almost completely collapsed. A stab wound was made on the 6th intercostal space and a pair of curved Spencer-Wells forceps were inserted through this wound into the right pleural cavity. A Malecot catheter was threaded through the opening in the diaphragm. The end of the Malecot catheter was grasped with the Spencer-Wells forceps and the catheter pulled out of the anterior chest wall. The catheter was connected to an underwater drainage bottle. The hole in the diaphragm was then closed with a layer of continuous No. 0 chromic catgut.

The left side of the diaphragm was similarly opened. This latter procedure was much easier, for the left lobe of the liver was not in the way. When the left pleural cavity was opened about 200 c.c. of blood and a considerable amount of air was found. The blood and air were sucked out and the pleural cavity was drained in the same manner as was done on the right side. The abdomen was closed.

As soon as the pneumothorax was relieved, the blood-pressure rose to 70 mm. Hg, and when the abdomen was closed the blood-pressure was 90 mm. Hg. As blood was found in the left pleural cavity and there was bilateral pneumothorax, it was decided to explore the neck wounds with a view to controlling the bleeding, and to close the tear in both domes of the pleura. Accordingly a collar incision was made over the lower part of the neck. After the investing fascia had been incised with the platysma, the left sternomastoid was first retracted, but, on finding that there was fresh bleeding deep to this muscle, it was divided completely. A piece of gauze was packed into the source of bleeding. The left scalenus anticus was found to be completely severed, but the left phrenic nerve was intact as it had already traversed the muscle in its course from the left to the right border of the muscle. The left transverse cervical vessels were cut across, but they had already thrombosed and were not bleeding. These were nevertheless ligatured. The gauze pack was now removed and slight oozing could be seen to come from the vertebral vein, which was cut across. The vein was caught and ligatured and further examination revealed that the thoracic duct had also been severed as it curved forwards and laterally to drain into the junction between the internal jugular and subclavian veins. The cut ends of the duct were ligatured with heavy silk. The tear in the domes of the pleura was caused by a stab wound that entered obliquely the left supraclavicular fossa. The instrument while transversing the lower part of the neck severed the scalenus anticus, the left transcervical vessels, the vertebral vein, and the terminal portion of the thoracic duct. By a miracle of some sort the knife missed the left innominate vein while it cut through both apical

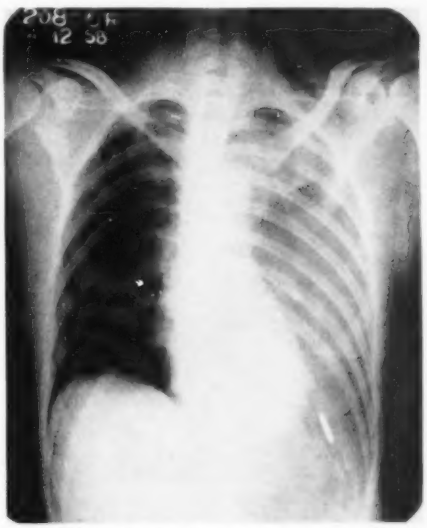

Fig. 568.—Radiograph of chest showing collapse of lungs with deviation of trachea.

pleura. The rent in the left apical pleura could be closed by atraumatic catgut and the neck closed without drainage. One pint of blood was given during the operation.

PROGRESS.—The patient returned to the ward in good condition. The blood-pressure by now had risen to 110/80 mm. Hg For the next two days there was good movement of the columns of water in the drainage tubes. Repeat radiography of the chest showed that there was still slight collapse of the right lung and a small amount of fluid in the left pleural cavity. The drainage tubes were removed on the third post-operative day. The general condition of the patient was excellent and she was out of bed and walking.

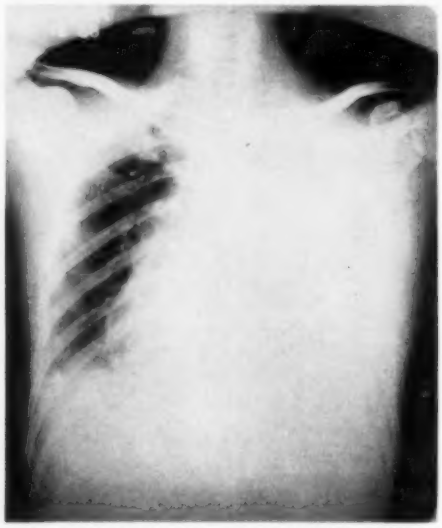

FIG. 569.—Showing total opacity of the left lung field.

On the fifth post-operative day she complained of shortness of breath and examination of the chest revealed diminished air entry in the left lung and there was dullness on percussion. Aspiration of the chest produced 200 c.c. of serosanguineous fluid with a certain amount of residual air. Radiography of chest (*Fig.* 569), done on the next day, revealed gross effusion on the left side. Aspiration was again carried out because of dyspnœa and 1500 c.c. of milky fluid was obtained.

1. *Examination of the Fluid.*—

a. Grossly it appeared milky with fat globules on the surface. It clotted after a few minutes of standing.

b. Microscopically. Fat globules were very evident. Cells: 980 W.B.C. per c. mm. Lymphocytes were the predominant cells.

c. Protein: 3·2 g.

d. Culture: No growth was obtained.

2. *Blood Examination.*—Hæmoglobin—72 per cent; R.B.C. 3,700,000 per c. mm. W.B.C. 9750 per c. mm. Neutrophils 9400 per c. mm. Cholesterol 100 mg. per cent; Protein Total 5·2 g. per cent; Albumin 3·2 per cent; Globulin 2·0 per cent.

SECOND OPERATION.—It was decided to explore the thoracic duct while her general condition was still good and accordingly on Dec. 16 the chest was entered through the left 6th intercostal space. On opening the pleura, about 1500 c.c. of chyle was sucked out. Clots of chyle which appeared like coagulated milk stripped off easily from the parietal and visceral pleura. The lung, which was bound down by the clotted chyle, became free and was expanding very well. Dissection for the exposure of the thoracic duct was then proceeded with by incising the mediastinal pleura above the aortic arch. The left subclavian artery was mobilized. The thoracic duct was found posterior and lateral to it. About 2·5 cm. from the origin of the left subclavian artery, a longitudinal tear measuring about 5 mm. in length was found in the thoracic duct. Clear fluid was seen to ooze out of it at a fairly rapid rate.

Attempts to close this tear with No. 00000 arterial silk were unsuccessful and so the duct was just freed and ligatured with heavy silk. The chest was closed with drainage. One pint of blood was given during the operation. The patient stood the operation very well and returned to the ward without any change in blood-pressure or pulse-rate.

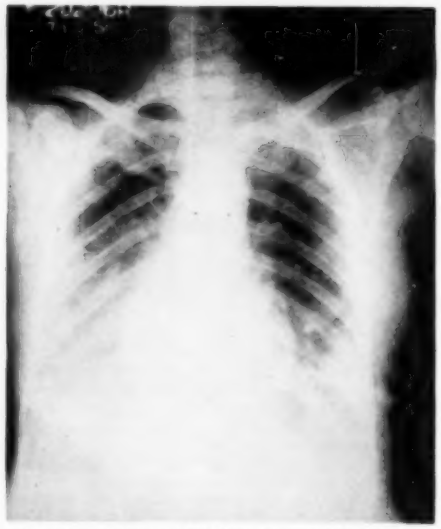

FIG. 570.—Radiograph of chest taken 3 days after transthoracic ligature of the thoracic duct, showing expanded lungs and clearance of opacity.

PROGRESS.—The convalescence of the patient was uneventful. The drainage tube was removed on the third post-operative day and she was able to walk about. Subsequent blood examination: Hæmoglobin 64 per cent; R.B.C. 3,000,000 per c. mm.; W.B.C. 5950 per c. mm.; Neutrophils 4750 per c. mm.

Radiography of chest (*Fig.* 570) showed that both lungs were expanded and there was no fluid present.

Her discharge from hospital was delayed until Jan. 5, 1959, in order to allow the Social Welfare Officer to help her in her social adjustment. Follow-up examination done on June 17 showed her to be well. The chest was clear, both on physical examination and radiologically. Blood examination:—Hæmoglobin 81 per cent; Hæmatocrit 41 per cent; R.B.C. 4,300,000 per c. mm.; W.B.C. 7350 per c. mm.; Cholesterol 130 mg. per cent; Protein 6·7 g. per cent; Albumin 4·5 g. per cent; Globulin 2·2 g. per cent.

DISCUSSION

There was an interval of five days between the original injury and the appearance of chylothorax. Two factors were probably at play in the production of the delayed rupture of the thoracic duct: (*a*) Localized weakness of the duct wall; (*b*) Increase of intraductal pressure.

The weakness could have been caused by the knife, having completely severed the thoracic duct at the root of the neck, grazing through the wall without penetration at the point of subsequent rupture. This weakness alone might not have caused chylothorax had it not been for the fact that there was also an increase of intraductal pressure.

The increase of the intraductal pressure was the result of ligature of the duct at the root of the neck at the first operation and oral intake of food. Crandall, Barker, and Graham (1943) showed that the flow of chyle was increased by oral intake of food especially if it contains fat.

It was felt that once chylothorax had developed it would be futile to treat the patient by aspirating the chyle and returning it by intravenous infusion, but that the duct should be ligated at the site of injury as soon as possible. Although Little, Harrison, and Blalock (1942) have given as much as 22 litres of chyle intravenously without any ill-effects, Whitcomb and Scoville (1942) have reported a case which died while intravenous chyle was being given. Meade, Head, and Moen (1950) suggested that the precipitation of fibrin might be the cause of the reaction and death. They therefore suggested that, if chyle were to be given parenterally, it should be filtered and given fresh. They were also against persistence in a conservative régime as coagulation of the chyle would cause pulmonary collapse and the loss of fat and protein would render the patient to such a poor nutritional state that surgery would become a very risky undertaking. Birt and Connolly (1952) reported a case which cleared up after repeated aspiration and phrenicectomy. They were, however, prepared to ligate the thoracic duct should this procedure fail.

Hodge and Bridges (1948), believing that simple ligature of the thoracic duct would cause increase of intraductal pressure and result in further rupture and chylothorax, managed to anastomose the duct to the vena azygos. Seaman (1954), having the same belief, repaired the tear in the thoracic duct by dissecting out a cone of pleura surrounding the opening. On closing the cone of pleura with a figure-of-eight suture, he succeeded in effecting a repair. Such complicated restorative procedures, in the opinion of Stranahan and others (1956), have not proved to be superior to simple ligature. In a series of studies of the thoracic duct by operative ductography, they showed that in at least a third of the cases the thoracic duct had two separate trunks at the level of the diaphragm, and concluded that it would be necessary to ligate the accessory ducts in order to ensure success. Shumacker and Moore (1951) considered that it was perfectly safe to ligate the thoracic duct because of the presence of collaterals.

Schaffner and Kirkpatrick (1951) advocated ligature of the thoracic duct through a right thoracotomy irrespective of whether the chylothorax was found in the right or the left side of the chest. This advice may be taken if there is difficulty in locating the damage in the thoracic duct. Klepser (1950), during a discussion on the paper presented by Meade and others (1950), reported a case in which he administered green coal-tar dye in olive oil and at operation obtained good visualization of the thoracic duct. Seaman (1954), utilizing milk and cream, could also locate the opening in the thoracic duct. This simple and effective method is easily obtainable and, although there was no necessity to use it in this case, it will certainly facilitate the localization of the point of leakage if it were administered by duodenal intubation a few hours before operation.

SUMMARY

A case of traumatic chylothorax resulting from stab wound of the neck is reported. The injury included opening of both domes of the pleura with bilateral pneumothorax. Ligature of the thoracic duct in the neck was followed five days later by chylothorax. It is postulated that the delay in appearance of chylothorax was due to weakness of the duct wall as a result of injury as well as increase of intraductal pressure. The increase in pressure was brought about by ligature of the duct in the neck and intake of food. A second operation undertaken to ligate the thoracic duct at the point of leakage cured the condition.

We wish to thank the Hon. Director of Medical and Health Services of Hong Kong for permission to publish this case and to make use of the records.

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A CASE OF PIGMENTED CONGENITAL EPULIS

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In 1926 Mummery and Pitts described a very rare, possibly unique, case of a pigmented congenital epulis. This was a maxillary tumour occurring in an infant of 6 months and on the basis of the histological finding regarded as a melanotic epithelial odontome.

Since that time some twenty similar cases have been recorded under a variety of designations, and located in differing sites.

These tumours, until recently, have usually been regarded as derived from the paradental epithelium,

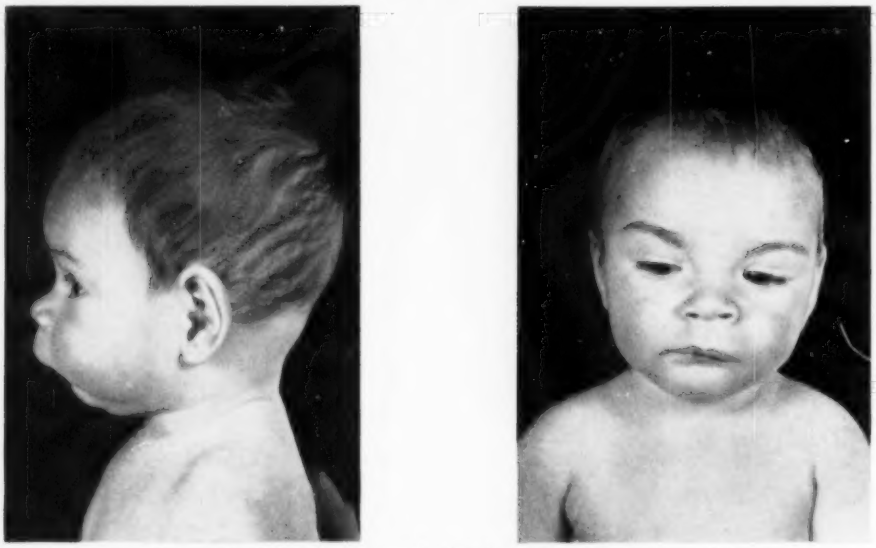

FIG. 571.—Photographs of the child aged 7 months when first examined, showing extent of the tumour.

FIG. 572.—Photographs of the child 2 months after operation, showing facial contour restored to normal.

and were therefore considered to be in the nature of adamantinomata. In 1947, however, Halpert and Patzner demonstrated cells resembling glia and advanced the theory that these maxillary tumours were derived from misplaced retinal or neural tissue, and the term 'retinal anlage tumour' was adopted.

In all, there are 21 cases in the literature, comprising 15 tumours of the maxilla, 4 of the mandible, 1 of the skull, and 1 of the epididymis.

The sex incidence shows a preponderance of females to males in the proportion of 15 to 6, and that universally it is a tumour of infancy occurring at an average age of approximately 6 months. The growth runs a benign course, even with incomplete excision. A further maxillary tumour is reported here.

CASE REPORT

A male infant aged 7 months was first seen at the Hospital for Sick Children in May, 1957, having been transferred from the Royal Berkshire Hospital who kindly forwarded the slides of a biopsy taken there.

The mother gave a history of a rapidly developing swelling in the upper incisor region of some five weeks' duration. The swelling was very apparent on examination, producing considerable deformity of the face, with bulging of the upper lip, alar base, and left cheek (*Fig.* 571). The tumour, extending from the right canine region to that of the left first molar, was found to be firm, smooth, and non-fluctuant. It was not tender on palpation, nor was there any ulceration of the mucosa. The teeth present were $\frac{BA|B}{}$. There was nothing relevant in the medical history, and physical examination, including the blood-picture, revealed no abnormality. The radiograph of the maxilla showed apparent expansion, although the nature and extent were difficult to demonstrate.

The histological slides accompanying the child were interpreted by one of us (M. B.) as follows:—

The tumour contained a stroma of fibroblastic tissue with whorl formation and in it there were two types of neoplastic cells:—

1. Small cells simulating neuroblasts with fibrillary processes arranged in clumps or rosettes.
2. Larger elongated cells which contained melanin and formed acini.

This tumour appeared to be a 'melano-ameloblastoma' or 'retinal anlage tumour' of maxilla.

On the basis of this report the tumour was considered non-malignant and suitable for conservative surgery.

At Operation (June 6, 1957).—An intra-oral incision was made over the tumour on the labial aspect of the alveolus from the $\underline{C|}$ region to the $\underline{|D}$. The mucosa, together with a tissue-paper thickness of the outer plate, was dissected from the tumour surface, the limits of which were then defined. The tumour was removed by blunt dissection, there apparently being no invasion of bone. During the operation two of the erupted deciduous teeth were unavoidably extracted, of which the $\underline{B|}$ remained attached to the tumour. The subsequent and comparatively large cavity left by the enucleation was packed with fibrin foam and thrombin, and the mucosal flap sutured into place. During the operation 150 c.c. of blood was administered.

The child was discharged from hospital after fourteen days, and made an uneventful recovery. When seen in March, 1959, there was no evidence of recurrence, and the facial contour was restored to normal (*Fig.* 572).

Biopsy (*Fig.* 573).—The tumour, which measured 3 cm. and 2 cm., was lobulated, firm in texture, and slate-grey in colour. Attached to the tumour was the $\underline{B|}$ and to one lobule was closely applied the crown of an unerupted tooth, presumed to be the missing $\underline{|A}$.

Microscopical Report (M. B.).—The bulk of the tumour stroma was young fibroblastic connective tissue showing a distinctly whorled arrangement. In it lay numerous gland-like acini and strands of heavily pigmented polygonal or strap-like elongated cells; the pigment was melanin. In addition there were minority areas of rather more undifferentiated smaller cells with deeply staining

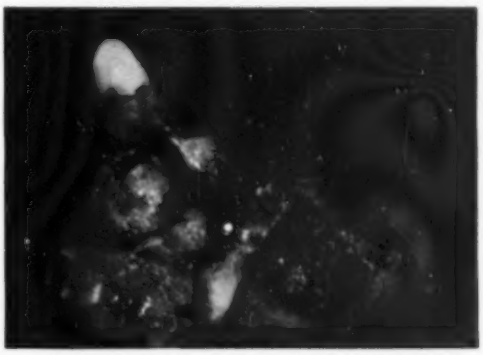

Fig. 573.—Showing the texture and melanin pigmentation found throughout the tumour. The deciduous lateral incisor is attached to one lobule of the tumour.

nuclei and fine fibrillary cytoplasmic processes forming a loose network. Special stains did not produce any evidence in favour of the neuroglial nature of these cells (*Figs.* 574–577).

The diagnosis was melanotic epithelial odontome.

DISCUSSION

The tumour described in this article bears a striking resemblance to the one so carefully documented and illustrated by Mummery and Pitts from the same hospital in 1926. In both cases a displaced deciduous incisor tooth was found in the growth and subsequent X-ray examination revealed the absence of the corresponding permanent tooth germ. But, whereas Mummery and Pitts were quite definite in their identification of primitive tooth elements, our findings do not correspond.

In 1958 Willis, who examined six tumours of this nature, was able to show evidence of included paradental epithelium in each one. He came to the conclusion that this was the origin of the tumour and not, as had been previously suggested by Halpert and Patzner (1947), heterotopic retinal tissue.

We have repeated the studies made by Professor Willis using special staining techniques in an effort to identify any normal or abnormal neuroglial or astroglial elements in the tumour, but with the additional advantage of having available frozen sections of tumour, whereas Willis had to base his conclusions on paraffin sections only. To this purpose we compared our tumour tissue with several controls including normal brain, a teratoma with ample areas of central nervous tissue, and an astrocytoma. While the controls produced good staining reactions with the appropriate stains, no such reaction was obtained with the sections of the tumour.

We therefore concluded that the hypothesis of the origin of these tumours from ectopic retinal tissue cannot be supported, nor can such a view be maintained on purely embryological grounds.

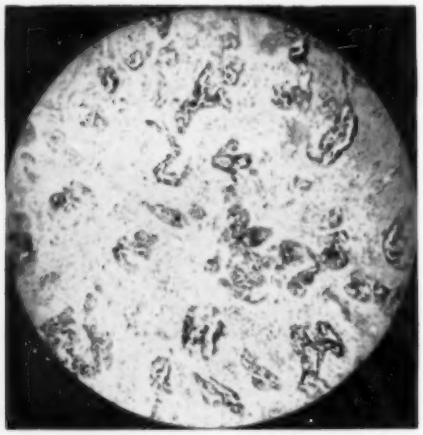

FIG. 574.—Photomicrograph × 54, showing gland-like acini of pigmented cells in a fibroblastic stroma.

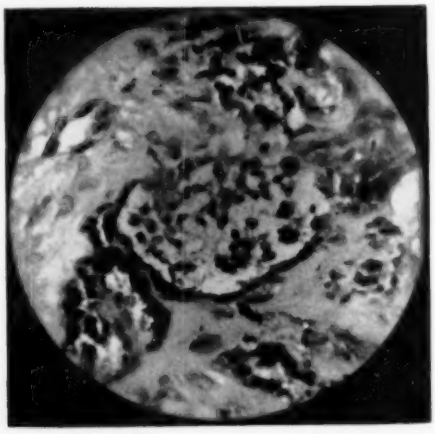

FIG. 575.—Photomicrograph × 275, showing the pigmented cells and an area simulating neuroglia.

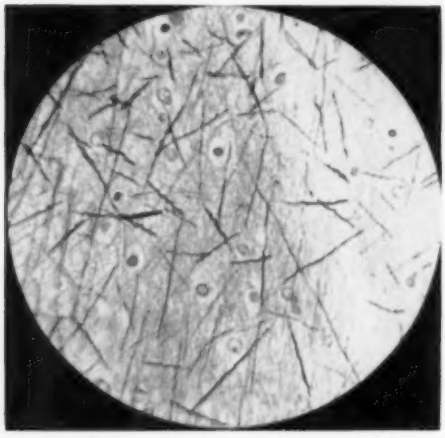

FIG. 576.—Photomicrograph of normal central nervous tissue stained for nerve fibres (silver stain).

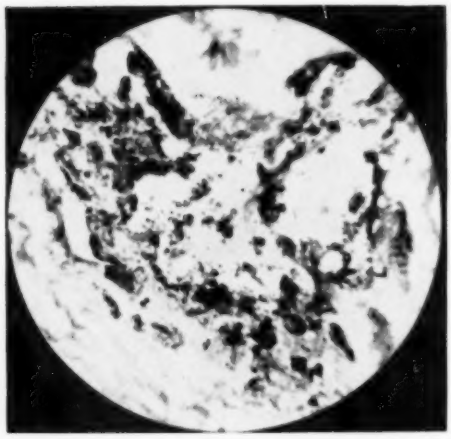

FIG. 577.—Photomicrograph of the tumour stained by the same method as the control. There are no nerve fibres, but the silver stained the melanin pigment.

Although we were unable to demonstrate any definite continuity of tumour cells with paradental epithelium in our case, such association has been amply demonstrated in other cases and we subscribe wholeheartedly to the views recently supported by Professor Willis that the pigmented epulis of infants is derived from retrogressing paradental epithelium with the appearance of melanin pigment in the cells. This benign lesion should therefore be considered as an odontomatous hamartoma rather than a true neoplastic process.

SUMMARY

A further case of a rare congenital epulis is described, with special reference to the histology.

It is considered that the tumour is not derived from primitive neural ectoderm, and is in the nature of an adamantinoma.

We acknowledge with pleasure the kindness of Mr. D. L. McKenzie, who first referred this case; and to Mr. D. Martin, for both the photography and photomicrography.

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HÆMANGIOMA OF THE BLADDER

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HÆMANGIOMA of the bladder is a rare condition, and the present paper is only the tenth reported case of the neoplasm occurring in a child. The first case on record was described by Arbuthnot Lane in 1895, at a meeting of the Clinical Society of London. This patient was a girl aged three and a half years for whom Lane performed a cystotomy for profuse hæmaturia, and "large nævoid masses, some as large as grapes, protruded through the incision". No attempt at excision was made and the bladder was closed. Unfortunately, it is not known what became of the child, although there was no further bleeding for the short period that she was followed up.

The presenting symptom in all cases has been hæmaturia. Diagnosis has been made in most instances at cystoscopy, though on several occasions no diagnosis seems to have been attempted until the bladder has been laid open.

The neoplasm takes one of two forms and the treatment differs according to the type. The simpler variety appears at cystoscopy as flat purplish patches, and responds well to fulguration. Macalpine (1930) describes such a case. The other type takes a more complex form, in which the tumour stands out from the bladder wall and varies in size from that of a cherry stone (Bachrach, 1910) to a mass 3 in. in diameter (Ballenger, Elder, and McDonald, 1932). Where the tumour has been at the smaller end of the scale, endoscopic fulguration has been carried out at first (Higgins, Williams, and Nash, 1951), but cystotomy and fulguration or partial cystectomy has been required sooner or later (Riches, 1951). Macalpine also describes the case of a boy who had had hæmaturia at intervals since birth. Cystoscopy showed a reddish-brown tumour in the midline posteriorly, which when the bladder was opened was seen to be half the size of a tennis ball. This tumour was excised.

In many patients there have been associated hæmangiomata on other parts of the body (Lane; Macalpine); there were no instances of malignancy.

CASE REPORT

The patient was a boy aged 12 years who had been attending the Hospital for Sick Children since 1952 for diabetes mellitus. The course and treatment of his diabetes proceeded satisfactorily until in December, 1957, he had several episodes of hæmaturia, having passed some clots on two or three occasions. An intravenous pyelogram showed no abnormality and he was admitted for investigation. In the two months that elapsed while awaiting admission there was no further hæmaturia. He had also an undescended testicle.

On Feb. 5, 1958, cystoscopy (J. P. P.) showed a small papilliferous growth high up on the left side of the bladder. It was a reddish-brown colour and stood out in thick fronds from the bladder wall. The tumour was thought to be a papilloma. The undescended testicle was mobilized and brought down into the scrotum.

Cystoscopy (D. I. W.) on Feb. 14, 1958, confirmed the findings of the previous examination. The tumour was too large to be treated endoscopically. The bladder was opened, revealing a dark red papilliferous tumour about 1 in. in diameter, attached to the bladder mucosa by a narrow base. There was no infiltration of the deeper tissues. The pedicle was clamped, the tumour excised, and the base oversewn. The bladder was closed, leaving an indwelling catheter.

PATHOLOGICAL REPORT (Dr. Bodian).—"The interpretation of this lesion has been somewhat difficult. On the whole, it presents as a papillomatous structure covered with transitional bladder epithelium which has deep

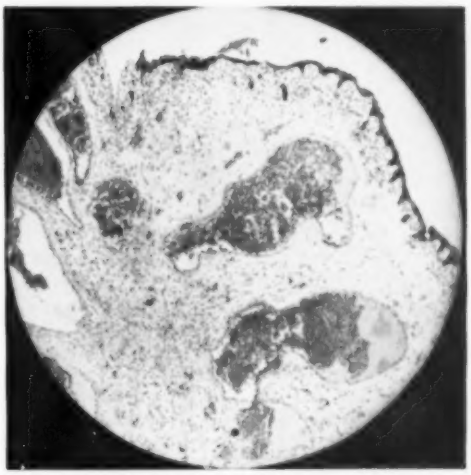

FIG. 578.—Photomicrograph showing numerous abnormal venous channels in the vesical submucosa. (× 37.)

ramified processes into the submucosa. These do not display any undue mitotic activity, but show some degenerative changes. The immediate submucosal tissue has a myxomatous structure and in it lie numerous large abnormal cavernous blood-vessels, some of which are thrombosed. Extravasation of blood must have occurred in the past and there are blood-pigment laden macrophages in the submucosa. There is no good evidence in the sections of any carcinomatous change nor of rhabdomyosarcoma, and I favour the diagnosis of a cavernous hæmangioma of bladder, producing a papillomatous appearance." (*Fig.* 578.)

I should like to thank Mr. D. I. Williams, under whose care the child was admitted, for permission to report this case, and for his great help in preparing this paper, and Dr. Bodian for the pathological report.

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POLYPOSIS OF THE SMALL INTESTINE, WITH PIGMENTATION OF THE FACE, BUCCAL MUCOSA, AND DIGITS

By J. OLDFIELD

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SINCE the initial description by Peutz in 1921 and the review by Jeghers and his associates in 1949, this interesting syndrome has been recognized and reported with increasing frequency. Richterich-van-Baerle (1956) reviews a total of 47 cases up to 1955 which were proven by operation, autopsy, or biopsy. Dormanby and Edwards (1956) investigated five families and 21 patients affected by this condition.

FIG. 579.—Marked pigmentation face, lips, and on dorsal aspects of interphalangeal joints.

The majority of cases give a characteristic story of recurrent central abdominal colic which has in many cases been sufficiently severe to require emergency laparotomy. The presence of the distinctive pigmentation and of intestinal colic would appear to be sufficient to justify the diagnosis.

CASE REPORT

A. B., a woman aged 27 years, was seen for the first time in the Out-patient Department of the Royal Victoria Infirmary, Newcastle upon Tyne.

She gave a history of two recent emergency operations at another hospital for acute abdominal pain. At the first laparotomy on April 27, 1951, she was thought to have a perforated peptic ulcer, but was discovered to have an ileo-ilial intussusception which was easily reduced. The cause was not discovered.

The second operation on March 29, 1952, for a similar attack showed an ileo-ilial intussusception with an ileal polyp at its apex. Reduction was carried out, but the patient's condition did not allow removal of the polyp.

In view of this story, and the fact that she was having recurrent mild attacks of intestinal colic, she was admitted for assessment and laparotomy.

ON EXAMINATION.—The patient was found to be a healthy young woman of good physique with marked freckling of the face, lips, and dorsal aspects of the interphalangeal joints (*Fig.* 579). The abdomen showed two recent laparotomy scars but no other abnormality.

Barium meal and follow-through using the Sprue technique showed "possibly one or two polyps in the small intestine. Demonstration not very convincing".

AT OPERATION (May 21, 1952).—Laparotomy was carried out through the old right lower abdominal paramedian incision. Several polyps were noticed throughout the small intestine. The first three were on the upper jejunum, the proximal one of these causing an intussusception which was still present. This segment of jejunum, approximately 12 in. long, was resected with end-to-end anastomosis. In the terminal ileum three more polyps were felt in a loop of bowel showing many adhesions suggesting previous intussusception. This was also resected.

FIG. 580.—*Upper specimen*: Jejunum showing numerous polyps, one large one causing partial intussusception. *Lower specimen*: Ileum showing diffuse distribution of small polyps.

The patient made a good recovery.

PATHOLOGICAL REPORT.—The pathologist, Dr. Schade, reported:—

1. The first specimen showed a length of ileum 27 cm., which had been opened longitudinally. On the mucosal aspect were numerous adenomata, the majority less than 0·5 cm., with three larger adenomata 2 cm., in diameter also present (*Fig.* 580).

2. The second specimen is a portion of jejunum 20 cm. long which had been inflated with formalin. On section the mucosal aspect showed numerous small and two larger adenomata, one of which had caused an intussusception which has reduced (*Fig.* 580).

Histology shows benign adenomatous polyps.

The patient was discharged home on June 3 and remained well until October, 1956, when she had an attack of abdominal distension which lasted three weeks. She had no pain and no nausea or sickness. The condition resolved and has not recurred. At the present time she is asymptomatic and her chief complaint is one of constipation.

Barium meal and follow-through showed "normal passage with no signs of polyposis".

DISCUSSION

The condition has been well documented and shows the salient features to be pigmentation of the face, buccal mucosa, and sometimes the digits, which may be present from birth or early childhood. The associated polyposis usually involves the small intestine, but the large bowel and stomach may be involved. In only 2 cases described were the polyps

confined to the colon. In Richterich-van-Baerle's (1956) review 57 per cent gave a definite family history. The present case would appear to be an isolated one, since her father shows facial pigmentation but has given no signs suggestive of polyposis. The patient's sister is unaffected.

The natural history of the condition seems to vary considerably in respect of the rate of growth of the polyps. One patient reported by Stewart and Storey (1955) had nine operative procedures between the ages of 6 and 16 years, ending with subtotal colectomy and ileorectal anastomosis. Most of these procedures were carried out on the colonic polyps. The condition in the small bowel appears to be more slowly progressive. The case reported has had no signs of recurrent intussusception since her operation seven years ago, and one must presume that none of the residual polyps have yet reached an adequate size to produce symptoms.

The incidence of malignancy appears to depend on the situation of the polyps. Richterich-van-Baerle's review gives an incidence of 15 per cent, which is considerably less than that found in familial polyposis coli, in which Lockhart-Mummery, Dukes, and Bussey (1956) report 154 of 218 affected cases developing cancer. Dormanby and Edwards (1956) question the incidence of malignancy and suggest that the invasiveness of the tumour is only apparent. Foster (1944) reported a father and daughter who presented with carcinoma of the small intestine.

The treatment is dependent on the areas of the bowel which are affected. If the condition is limited to the small bowel the surgical procedures must of necessity be conservative, and limited resections or removal of the polyps through multiple enterotomies would appear to be the wisest course. Extensive polyposis of the colon with the known tendency to early malignant degeneration will require a more radical approach as advocated in familial polyposis coli.

SUMMARY

A case of polyposis of the small intestine with pigmentation of the face, buccal mucosa, and digits is presented.

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REVIEWS AND NOTICES OF BOOKS

L'Arteriopatia Ostruttiva Aorto-Iliaca. By ENNIO MUNTONI, J. DEL MORO, R. LISCIA, and F. PIZZETTI. 1959. Firenze: Ospedale di S. Giovanni di Dio. 9¼ × 6½ in. Pp. 185, with 84 illustrations, 16 in colour. L.3500.

TWENTY years ago, application of the then comparatively recent technique of peripheral arteriography was yielding important new knowledge. One of the lessons of that epoch was the discovery that obstructive lesions may often be much more proximally sited than the symptoms to which they give rise. It is not surprising, therefore, that the newer method of aortography provides further data illustrating the same principle. The present monograph is based on the careful study of over 70 patients with obliterative disease of varying extent and degree in the aorta and iliac vessels, including 6 with Leriche's syndrome of complete obstruction of the lower aorta. In addition to the presenting symptoms of limb ischæmia, very many of these patients also had visceral disturbances of the kind sometimes called 'abdominal claudication'. Aortography shows that as the aorto-iliac obliterative process advances, the superior mesenteric artery, colic arcade, and (so long as it is not itself obliterated at the source) inferior mesenteric artery undergo great dilatation and through the hæmorrhoidal plexus provide important collateral sources of arterial blood for the limb. It seems probable that these hæmodynamic changes also affect the circulation of the gut wall, so contributing to the symptoms. At operation in such cases there is always a marked degree of periarteritis extending into the root of the mesentery, and the authors consider that this, too, by causing irritation of the visceral autonomic nerves, plays a major part, while direct involvement of the mesenteric vessels by the disease process accounts for a further increment. The combination of abdominal and lower-limb symptoms seems, however, to be characteristic of arterial disease in this situation. Other cases show involvement of the renal circulation with associated hypertension.

The effects of obstruction in various situations are considered in great detail and illustrated by radiographs which have been skilfully re-touched to give an effect of startling clarity, in appearance resembling something between a fossil and a bas-relief. Further chapters are devoted to the syndrome of primary dolicho-mega-artery (described by Leriche), which is critically considered and not unreservedly accepted; peripheral neuropathy (objective changes in 40 per cent); and aetiopathogenesis, in which, besides much that is familiar to English-speaking readers, reference is made to a considerable body of recent Italian work on blood chemistry, including studies of the clotting mechanism, albumin, globulin, lipo- and glyco-protein fractions, and plasma metachromatic (heparinoid) bodies.

The final chapter deals with surgical treatment. Neither sympathectomy alone nor excision of the aortic bifurcation is recommended and the choice lies between thrombo-endarterectomy and arterial grafting. The former operation has lately come under a cloud, but the authors point out, firstly, that it should not be confused with simple disobliteration, which is an inferior procedure; and, secondly, that results are much better in large arteries such as the aorta and iliac vessels than in others of lesser calibre. Several excellent results followed for a number of years seem to bear out this assertion, and the method is recommended in cases where the obstruction

is segmental and wholly above the femoral vessels, especially if the patient is young. Obliteration of the aorta extending right up to the renal arteries is a further indication. Otherwise, the authors now use lyophilized homografts, and on the basis of the past two years' experience considers these to be satisfactory except for very extensive replacements (aortic bifurcation to adductor hiatus), when a nylon prosthesis is preferred.

The text is followed by a useful and accurate bibliography listing over 200 European, British, and American books and articles, with titles as well as authors and sources. Printing, illustrations, and binding are excellent, and the work is a valuable original contribution to its subject.

Osteochondritis Dissecans. Loose Bodies in Joints: Etiology, Pathology, Treatment. By I. S. SMILLIE, O.B.E., Ch.M., F.R.C.S.(Ed.), F.R.F.P.S., Lecturer-in-Charge, Department of Orthopædic Surgery, University of St. Andrews. 9½ × 6½ in. Pp. 224 + viii, fully illustrated. 1960. Edinburgh & London: E. & S. Livingstone Ltd. 60s.

THIS monograph is devoted to the aetiology, pathology, and treatment of loose bodies in joints. Although the condition has been described for over two hundred years, yet it is only since the extensive use of X rays that it has received attention by orthopædic surgeons on both sides of the Atlantic. Mr. Smillie, who is well known for his book on *Injuries of the Knee Joint*, has now presented us with an equally useful volume on the subject of osteochondritis dissecans, a condition which is much more common than was formerly thought.

The juvenile variety is encountered towards the second decade, and is most frequently met with in the knee-joint; it originates in an anomaly of ossification or abnormality of endocrine function. Rarely the condition may be seen in more than one member of a family.

The author describes the various operations which are practised to-day in the treatment of this condition. Although removal of loose bodies is generally performed, internal fixation of plaques of bone with vitallium pins or screws appears to give good results. The illustrations, especially the coloured plates, are excellent and render the book all the more valuable as a reference volume.

The Year Book of General Surgery 1959-1960. Edited by MICHAEL E. DE BAKEY, B.S., M.D., M.S. 7¾ × 4¾ in. Pp. 639, with 169 illustrations. 1960. Chicago: The Year Book Publishers Inc. (London: Interscience Publishers Ltd.). 60s.

THIS hardy annual covers the whole aspect of general surgery and gives the reader a good idea of surgical progress. The editor is to be congratulated on the way he has chosen the more important papers which have been published during 1959 and commented upon them.

Cardiovascular surgery has come to the fore again and the artificial heart–lung apparatus is being more widely used.

Transplantation of splenic or marrow cells is now being carried out in many places in the world and the papers abstracted in this book are excellent.

This book is a useful guide to what is new in surgery and it should have a wide appeal.

The Surgical Treatment of Scoliosis. By LOUIS A. GOLDSTEIN, M.D., F.A.C.S., Associate Clinical Professor of Orthopædic Surgery, University of Rochester Medical Center, U.S.A. 9¾ × 6½ in. Pp. 116 + xii, with 47 illustrations. 1960. Oxford: Blackwell Scientific Publications. 54s.

SCOLIOSIS is always a difficult condition to treat and there is no quick cure. This monograph is based upon the personal experiences of the author and his matured clinical study covers a number of years, nearly a quarter of a century.

The author describes the technique of application of the turnbuckle plaster cast and gives a very detailed description of the more important points and includes several excellent clinical photographs.

The operation for spinal fusion is well described and illustrated.

Thirty-one clinical cases are illustrated and the pre- and post-operative radiographs are included.

This is a very comprehensive monograph on the surgical treatment of scoliosis and it contains an extensive list of references.

Dr. Vernon Thomas contributes an appendix on anæsthesia in scoliosis.

Die Wirbelsaulenleiden und ihre Differentialdiagnose. By J. E. W. BROCHER. 11 × 7¾ in. Pp. 457 + xvi, with 287 illustrations. 1959. Stuttgart: Georg Thieme Verlag. D.M.128.

THE theme-song of this book, now in its second edition, is early radiological diagnosis of non-traumatic spinal affections, the object being the eradication of such local conditions as classical Pott's disease and advanced ankylosing spondylitis. But the scope of the work, part of an atlas devoted to the radiology of normal and abnormal anatomy, goes far beyond its title, except where general diseases involving the spinal column are concerned.

Commencing with pathogenesis and ending on therapy, the subject-matter is fully reviewed: the result is almost as much a clinical as a radiological exposition, rendered remarkably comprehensive by the beautifully illustrated case histories which enrich the text.

This outstanding contribution from Switzerland deserves a wider public than that imposed by the language barrier.

Le Nouveau-Né. By MARCEL FÈVRE. 9¾ × 6 in. Pp. 200, 28 illustrations. 1959. Paris: G. Doin et Cie.

THIS volume of some 200 pages octavo of rather large print is one of a *Therapeutic Library* of which some forty other volumes are either available or in preparation. A glance at some of the other titles suggests that if the volumes are of equal size the subjects are probably treated in much greater detail than is the case here.

In British pædiatric surgical practice the phrase 'neonatal surgery' has a precise meaning which is not followed in this work in which such subjects as hare-lip, hypospadias, ectopia of the bladder, and congenital dislocation of the hip, amongst many others, are discussed. It is, indeed, rather a description of the surgical conditions of the neonatal period and of early childhood, with a discussion of the indications for treatment and prognosis rather than a work on the minutiæ of neonatal surgical practice.

An introductory note explains that the volume is intended primarily to inform the general practitioner about the lesions he may find at birth and in early infancy, so that he may discuss their management and prognosis intelligently with the anxious parents. This purpose is admirably fulfilled.

Les Entretiens de Bichat 1959. Chirurgie-Spécialités. 8$\frac{7}{16}$ × 6½ in. Pp. 503. 1959. Paris: Expansion Scientifique Française.

THE 1959 *Entretiens de Bichat* which took place at the Salpêtrière, October 4–11, formed part of the Paris Medical Week, together with a Scientific Exhibition and a series of Medical Films, Television Demonstrations, and so forth. The present volume, containing the papers delivered at the Surgical Section, was printed in advance

for the benefit of those attending and now serves as a record of the proceedings. It contains 104 papers, mostly very short, dealing with a great variety of topics in the fields of General and Special Surgery. These appear to have been intended for a wide audience rather than a gathering of specialists, and for the most part they provide rapid surveys of familiar topics, usually stressing some particular point which the author considers to be specially important. One of the few exceptions to this generalization is a very brief communication by Dargent, reporting the treatment of advanced breast cancer by adrenalectomy followed by an intrasplenic adrenal graft. This diverts the adrenal hormones to the portal circulation, where it is believed that the œstrogens are inactivated, while the mineralocorticoid and glucocorticoid fractions are not.

There are no illustrations and none of the articles has a bibliography. The book is well printed, well indexed, and well bound in cloth boards.

Outline of Orthopædics. By JOHN CRAWFORD ADAMS, M.D.(Lond.), F.R.C.S.(Eng.), Consultant Orthopædic Surgeon, St. Mary's Hospital, London. Third edition. 8½ × 5½ in. Pp. 440 + viii, with 313 illustrations. 1960. Edinburgh and London: E. & S. Livingstone Ltd. 35s.

THAT a third edition of this student's manual of orthopædics should be required just four years after the first edition speaks for the popularity of this book.

It is just what the student requires; basic orthopædic surgery, well illustrated with a lucid but dogmatic text. An excellent student's handbook.

Instructional Course Lectures. Volume XVI, 1959. Edited by FRED C. REYNOLDS, M.D. 10¾ × 8¼ in. Pp. 335, with 260 illustrations. 1959. St. Louis: The C.V. Mosby Co. (London: Henry Kimpton). £6.

THIS volume of instructional course lectures contains many interesting articles on athletic injuries, rehabilitation in athletes, the surgery of the paralytic hand, and internal derangements of the knee.

It is interesting to note that the 1959 International Course Program consisted of 124 courses presented by 181 instructors and was attended by 1700 physicians—which was again two-thirds of those registered at the meeting of the American Academy of Orthopedic Surgeons.

The articles dealing with femoral head prosthesis are especially good. The specific indications for the use of these prostheses are still not clear-cut, but the extended use must in time give us useful information.

This is the sixteenth volume of lectures and the whole series form an excellent library of orthopædic literature. We look forward to reading more volumes in the future.

The Foot and Ankle. By PHILIP LEWIN, M.D., F.A.C.S., F.I.C.S. Fourth edition. 9¼ × 5¾ in. Pp. 612, with 339 illustrations. 1959. London: Henry Kimpton. 105s.

THE fourth edition of this well-known monograph on the surgery of the foot and ankle is most comprehensive; it covers the injuries, diseases, deformities, and disabilities of the foot and ankle. The author, with his forty years' clinical experience, has a rich surgical background from which to draw while writing this edition.

Trauma plays an ever increasing part in orthopædic problems to-day and the stabilization of the injured foot is a major problem. Dr. Lewin is at his best in setting out the treatment of ankle injuries, and his attempts to tell the reader what to do, when to do it, and what not to do, are very rewarding.

The illustrations throughout the book are excellent, and, what is very refreshing, the majority of the drawings are original.

Cancer. Volume 6. Edited by RONALD W. RAVEN, O.B.E.(Mil.), T.D., F.R.C.S., Joint Lecturer in Surgery, Westminster Medical School, University of London. 9¼ × 6¼ in. Pp. 500 + x, with 138 illustrations. 1959. London: Butterworth & Co. Ltd. 85s.

WITH the appearance of Volume 6, this great work is brought to a conclusion. It may be said that this volume is one of the best. More than half is concerned with Chemotherapy, and an excellent account of the experimental background sets the tone. Hormone studies in breast cancer are dealt with in a masterly fashion by Bulbrook and Strong, whose exceptional experience in this field gives to their chapter great authority. Adrenalectomy and hypophysectomy for breast cancer have insinuated themselves rather surprisingly under the title of Chemotherapy, but not inappropriately so. As in some other sections of this work, no real advantage seems to have been gained by the annexation of transatlantic writers where the home product would have done as well or better.

The latter half of the volume deals with the Public Health and nursing aspects of cancer, and a final chapter by Haddow on the organization of cancer research winds up the discourse.

The editor and publishers are to be congratulated on a great contribution to British Medicine.

The Treatment of Bronchial Neoplasms. By ROBERT R. SHAW, M.D., DONALD L. PAULSON, M.D., and JOHN LESTER KEE, jun., M.D. 9¾ × 6¼ in. Pp. 135 + xv, fully illustrated. Springfield, Illinois: Charles C. Thomas (Oxford: Blackwell Scientific Publications). 64s.

THIS monograph deals with an important problem which is facing the medical profession both at home and abroad, because cancer of the lung is on the increase.

The authors of this book have made use of vast clinical material, including 1180 patients with cancer of the lung. There are few centres which can survey such an enormous collection of cases.

Perhaps the best part of this book is devoted to considering which lesions are suitable for surgery and which will be benefited by radiotherapy. The use of chemotherapeutic methods is also discussed.

Mr. John Kee has written a very interesting chapter on bronchial adenoma which is quite comprehensive and is the best account so far published on this lesion.

This monograph is profusely illustrated with excellent pictures and radiographs. A very useful production.

Pediatric Neurosurgery. Edited by IRA J. JACKSON, M.D., M.Sc., F.A.C.S., F.A.C.A., and RAYMOND K. THOMPSON, M.D., F.A.C.S. 10 × 6¾ in. Pp. 564 + xvi, fully illustrated. 1959. Oxford: Blackwell Scientific Publications Ltd. 125s.

THERE can be little doubt that the time is ripe for a comprehensive survey of pædiatric neurosurgery and Jackson and Thompson have presented us with a book which gives us just that review.

Neurosurgery is one of the newer branches of surgery and the two world wars have extended its influence considerably.

In this volume the editors have collected twenty-four contributors who write from matured clinical experience on every aspect of neurosurgery; these include pædiatricians, anæsthetists, neurologists, pathologists, and plastic surgeons.

The whole, modern, enlightened field of neurosurgery is incorporated in this book. The diagnostic side of neurosurgery, with all the up-to-date tests and clinical investigations, is well described in a very lucid way. The illustrations are excellent, especially the radiographs.

The surgery of epilepsy is admirably described.

The role of the general practitioner is not forgotten, for it must be remembered that it is he who first sees the little patient with the neurosurgical lesion.

A stimulating and practical book full of clinical knowledge.

Anatomy, Regional and Applied. By R. J. LAST, M.B., B.S.(Adel.), F.R.C.S., Professor of Applied Anatomy, Royal College of Surgeons of England. Second edition. 9½ × 6¼ in. Pp. 741 + xvi, with 372 illustrations, many in colour. 1959. London: J. & A. Churchill Ltd. 80s.

IT is always difficult to write a new text-book of anatomy because there are such good well-established ones in Great Britain. However, in 1954 Professor Last felt, quite rightly, that a new approach was necessary to the teaching of anatomy and so he produced his book, which immediately became popular amongst postgraduate students. The first edition was reprinted two years later and now a second edition has been called for.

There can be no doubt that this book is born of popular demand, and the second edition which has been carefully revised, and to which many more illustrations have been added, should prove of real help both to undergraduate and postgraduate students.

There is a very useful list of anatomical names now in use, together with the new ones which are generally used both by surgeons and anatomists. What the International Congress of Anatomists which is being held this year in New York will provide in the way of a new nomenclature remains to be seen.

We congratulate Professor Last on his continued success.

Cancer of the Breast. Compiled and edited by WILLARD H. PARSONS, M.D., F.A.C.S. 9 × 6 in. Pp. 232 + xvi, illustrated. Oxford: Blackwell Scientific Publications Ltd. 60s.

THIS is a stimulating monograph dealing with an all too common disease. The editor has collected eleven contributors who have covered every aspect of cancer of the breast. There are interesting discussions on the more controversial aspects of the disease. Dr. Parsons himself contributes an excellent chapter on "Present Status of Carcinoma of the Breast" and this sums up the surgical consensus of opinion in England to-day. The treatment of early cases of breast cancer is radical mastectomy.

The Royal Edinburgh Hospital for Sick Children 1860–1960. By DOUGLAS GUTHRIE. 8¾ × 6 in. Pp. 75 + xii, with 34 plates. 1960. Edinburgh & London: E. & S. Livingstone Ltd. 17s. 6d.

THIS is a delightful account of a Children's Hospital which has enjoyed an international reputation for well over sixty years. Its life history of one hundred years is told in a fascinating way by a well-known medical historian who has received the help of some contributors each writing on a special topic. It is noteworthy that since 1863, when Queen Victoria gave her gracious permission to the addition of 'Royal' to the title of the Hospital and conferred her Patronage upon the institution, the Hospital has enjoyed the privilege of the continued interest of members of the Royal Family. This Royal Patronage is no mere formality, and this is shown by the large number of Royal Visits during the century of the hospital's existence.

Many distinguished medical men have been on the staff of this hospital, but perhaps the two names that stand out are those of Sir Harold Stiles and Sir John Fraser, and these two between them served the hospital for some sixty years, surely a great achievement.

This volume is beautifully illustrated throughout and the coloured frontispiece of Her Majesty Queen Elizabeth, the Queen Mother, is a lovely and attractive picture. All those who are interested in medical history, and there are many, will find this book a pleasure to read. The price of the book is most moderate and this is quite uncommon to-day.

Metals and Engineering in Bone and Joint Surgery. By CHARLES ORVILLE BECHTOL, M.D., ALBERT BARNETT FERGUSON, jun., M.D., and PATRICK GOWANS LAING, M.B., B.S., F.R.C.S. 9⅛ × 6½ in. Pp. 210 + viii, with 123 illustrations. 1960. London: Baillière, Tindall & Cox. 64s.

THIS monograph incorporates a wealth of material which would be difficult for a surgeon to obtain except from a study of many books on metallurgy, physics, and dynamics. It is interesting to note that Lord Lister was one of the first surgeons successfully to use buried silver wire sutures in the internal fixation of fractures. And it was Hey-Groves who first made a detailed study of tissue tolerance to metals in 1913, and he reported on the use of an intramedullary bar for comminuted fractures.

This book deals with corrosion of metals within the body, more especially in bones, with fatigue fractures of metals, and with many other aspects of metal behaviour.

Surgeons who are in the habit of using metal plates, screws, nails, bands, or wire would do well to consult this book. A valuable reference volume.

Thérapeutique Chirurgicale des Ictères par Rétention. By P. GOINARD and G. PÉLISSIER. 9½ × 6¼ in. Pp. 210, with 82 illustrations. 1959. Paris: Masson. 3200 fr.

ONE feature of surgery in post-war France is the considerable interest there in diseases of the biliary tract. This monograph reflects this interest—it is the first, in fact, to deal exclusively with the important problem of the management of obstructive jaundice.

The book is divided into two parts: The first deals with the pathology, cholangiography, and principles of surgical management in jaundice due to stone, operative injury, chronic pancreatitis, malignancy, congenital obstructions, and the more exotic causes such as parasitic diseases.

The second section deals, quite briefly, with the techniques of surgical intervention. It is particularly interesting that the authors advocate the removal of common-duct calculi via a widely incised sphincter of Oddi when the exact number and situation of the stones can be determined with certainty by cholangiography; the common duct is left intact and undrained.

Apart from this somewhat controversial point, there are few features with which British surgeons will disagree.

Once again, however, one is sad to see that this country is but poorly represented in the numerous references; an indication of the barrier that still exists between Continental surgeons and ourselves.

The Surgical Treatment of Facial Injuries. By VARAZTAD HOVHANNES KAZANJIAN, M.D., D.M.D., and JOHN MARQUIS CONVERSE, M.D. Second edition. 10 × 6½ in. Pp. 1110 + xi, with 1115 illustrations. 1959. London: Baillière, Tindall & Cox. 256s.

THE second edition of this large and comprehensive work on facial injuries appears some ten years after the first. During that decade much has happened to plastic surgery and to the treatment of facial injuries. Plastic surgery may be said to have been born during World War I, to have become of age between the wars, and to have grown

to manhood during World War II. The two world wars gave the plastic surgeons a vast experience and new techniques and operations were devised.

We are living in an age when traumatic surgery is on the increase and plastic surgery plays a large part in this increase. This new second edition has in part been rewritten and six new chapters have been added. These new chapters enhance the value of the book and include: Introduction to Fractures of the Facial Bones; Fractures of the Fronto-ethmoidal Region and Traumatic Cerebrospinal Rhinorrhœa; Facial Injuries in Children; X-ray Examination of the Facial Bones; the Transplantation of Tissues; and Scars of the Face.

Over 400 new illustrations have been incorporated in this edition, and some of these are in colour.

Burns are all too common to-day in spite of many precautions which have been instituted in the home and in the factory. The plastic surgeon deals with these cases from the start and better results are being obtained. Burns of the face are a problem in themselves and this problem is ably considered in this book.

This important work reveals the importance of team work in treating facial injuries; the dental surgeon, the ophthalmic surgeon, and ear, nose, and throat surgeon all play their part with the plastic surgeon in the treatment of facial injuries. As one would expect in a large work such as this, the bibliography is extensive, and it is also accurate.

Every surgeon who treats cases of facial injury should obtain a copy of this work.

Chirurgie der Verengungen der Oberen Luftwege. By AUREL RETHI. 9½ × 6½ in. Pp. 119 + viii, with 116 illustrations. 1959. Stuttgart: Georg Thieme Verlag. D.M.28.

THIS is a monograph on the subject of congenital, post-inflammatory, post-operative, and neurological atresias or stenoses, written by a laryngologist who has specialized in the plastic problems of the upper respiratory tract.

After discussing certain general principles of plastic surgery, the book deals in an anatomical manner with stenosis as it occurs in the various portions of the upper air-passages. The subject is well covered and the operative procedures are often those of the author's personal preference. The illustrations are very numerous and carefully and clearly executed. At the end of the booklet there are 7 pages of references, but only a few deal with post-war publications.

Surgery of Repair as applied to Hand Injuries. By B. K. RANK, C.M.G., M.S.(Melb.), F.R.C.S. (Eng.), F.R.A.C.S., Hon. Plastic Surgeon, Royal Melbourne Hospital; and A. R. WAKEFIELD, M.S.(Melb.), F.R.C.S.(Eng.), F.R.A.C.S., Plastic Surgeon, Royal Children's Hospital, Melbourne. Second edition. 9½ × 6½ in. Pp. 284 + xiv, with 219 illustrations. 1960. Edinburgh and London: E. & S. Livingstone Ltd. 45s.

THIS is a second edition of a text which has already been widely acclaimed as a most important contribution to the ever widening field of traumatic surgery. The authors have made only minor changes and have wisely resisted any impulse to expand the subject beyond the confines of their title. None the less it must be made clear that this book is not only an extremely well-written and valuable contribution to a specialized surgical field, but is also, as the title suggests, an essay in the surgery of repair. The authors have presented the fruits of their own very considerable experience, and have not attempted to encompass all the alternative methods of treatment. This has made for a clarity and continuity of text which is wholly admirable, making the book most readable. The work is well illustrated, some of the results shown bearing witness to the very high standard of skill which has been set by these two distinguished Australian surgeons.

Of particular interest are the chapters on the surgical anatomy of the hand, in which a reassessment of the important anatomical features is made in the light of the shift of emphasis from major hand sepsis to trauma. Also of general interest are the chapters on the social significance of hand injuries; first-aid methods; the examination of and approach to the recently injured hand and its primary treatment; and the care of the burnt hand.

This is not merely a book for the specialist, but could be read with profit by all surgeons. Those concerned specifically with the treatment of hand injuries can hardly afford not to have it on their shelves.

Metabolic Homeostasis. By NATHAN B. TALBOT, M.D., ROBERT H. RICHIE, M.D., JOHN D. CRAWFORD, M.D., and EDITH S. TAGRIN. 9 × 6¾ in. Pp. 132 + xii, fully illustrated. 1960. Published for the Commonwealth Fund by Harvard University Press (London: Oxford University Press). 24s.

THE metabolic needs of patients have in the last fifteen years received more intensive study than at any other time in history. The elaboration of isotopic tools and advances in physical chemistry have improved greatly our knowledge of metabolic processes. Matching this advance, numbers of books have been published ranging from those which require a lectern to read comfortably, and are for the edification of the expert, to slender manuals for the enlightenment of the student. Amongst the latter class this book must take an important place. Little more than a pamphlet with pages on ring fastenings, it is just too big to go into the pocket; nevertheless, all the information necessary for the practising doctor or the inquiring student is contained within its pages.

Section A deals with the scientific aspects of water and electrolyte metabolism and each section is profusely illustrated with excellent line diagrams which serve to illuminate the argument in a most satisfactory manner. Each of these sections ends with a table listing the prominent symptoms and signs, on the one hand of depression, and on the other hand of excess, of the electrolyte in question.

Section B deals with the clinical application of the principles enunciated in the former sections.

The authors, being pædiatricians, emphasize throughout the particular needs of children at various ages, and the section on Burns provides a valuable chart illustrating the contributions of various parts of the body to the total surface area according to age. This chart must be most valuable in the treatment of burns in children.

On the whole this slender volume may be thoroughly recommended as a reasonably comprehensive, admirably clear, and eminently practical contribution to this important subject.

Atlas Aide-Mémoire d'Anatomie. By H. ROUVIÈRE. Second edition, revised by G. CORDIER. 10 × 7½ in. Pp. 336, with 241 illustrations. 1959. Paris: Masson et Cie. 4000 fr.

IN his preface to the first edition of this atlas the late Professor Rouvière wrote: "Of the facts that have to be learned in the course of the study of anatomy the majority fade from the memory, but a word or a picture alone may suffice to bring them back. I hope that this book will be helpful to the doctor or student who has learned human anatomy and has forgotten it all." It is in this spirit that the second edition has been prepared by Gaston Cordier, Henri Rouvière's successor as Professor of Anatomy in the University of Paris.

The book is arranged regionally; there has been little change, and most of the original illustrations remain.

The text has been kept as concise as possible, and there is a good index.

It has, however, been found necessary to add illustrations in those regions where later researches have demanded them, notably in the inner ear, the pericardium, and the lungs. In the case of the last-named there is effective economy of illustration and description, four blocks from Professor Cordier's own monograph on pulmonary anatomy neatly outlining the salient features. It still seems a widespread practice among anatomists to depict pulmonary arteries red and pulmonary veins blue. It is also surprising that the internationally agreed nomenclature of the pulmonary segments (1949) is not more widely used; but in Great Britain we can take pride in the fact that in most other European countries the apical segment of each lower lobe is named after H. P. Nelson, a great pioneer in pulmonary anatomy as well as in pulmonary surgery.

The book, though paper-backed, is well produced on high quality paper. The simplicity and clarity of its original plan should ensure its continued success. It is only fair to add that its cost is quoted in 'old' francs.

Management of the Aged Surgical Patient. By SIDNEY E. ZIFFREN, M.D., Professor of Surgery, State University of Iowa College of Medicine. 8¾ × 5½ in. Pp. 219, with 30 illustrations. 1960. Chicago: The Year Book Publishers Inc. (London: Interscience Publishers Ltd.) 57s.

GERIATRIC surgery has come to stay as the number of aged people in the world increases each year. On the whole, as this book shows, the aged person stands operative intervention remarkably well, especially if there has been good pre-operative preparation. Operations of the calibre of radical mastectomy in patients over 80 years of age show a remarkably small mortality; the same may be said of cases of partial colectomy. With modern anæsthesia, intravenous fluids, including blood transfusion, and a good pre-operative preparation elective surgery can be carried out with a small mortality in the over eighties.

Fundamental Techniques of Plastic Surgery and their Surgical Applications. By IAN M. MCGREGOR, M.B., F.R.C.S.(Eng.), F.R.F.P.S.(Glas.), Consultant Plastic Surgeon, Glasgow Royal Infirmary. 8⅝ × 5½ in. Pp. 244 + viii, fully illustrated. 1960. Edinburgh & London: E. & S. Livingstone Ltd. 30s.

THIS is a really useful book and one which will find popularity not only with the postgraduate but also with those surgeons who have to deal with the results of trauma to the human frame.

The book, a model of what a monograph should be, is divided into two parts. The first part is concerned with basic techniques and these have been admirably described and illustrated. Part Two deals with surgical applications as may be met with in general surgery, orthopædic surgery, hand surgery, and surgery of the eyelids.

There is a very definite need for such a book as this and Mr. McGregor is to be congratulated on a first-class achievement.

Professor Illingworth, who writes a foreword to this book, says: "It will assuredly receive a warm welcome", and we certainly believe it will.

The price of the book is very moderate.

BOOK NOTICES

[The Editorial Committee acknowledge with thanks the receipt of the following volumes. A selection will be made from these for review, precedence being given to new books and to those having the greatest interest to our readers.]

Chemical Mechanisms in the Induction of Cancer. By ALEXANDER HADDOW, M.D., D.Sc., F.R.S. No. 4 of Lectures, Monographs and Reports, 1959, of the Royal Institute of Chemistry. 9¾ × 6 in. Pp. 20 with 5 figures. 1960. London: The Royal Institute of Chemistry. 4s.

Klinische Chirurgie für die Praxis. Edited by O. DIEBOLD, H. JUNGHANNS, and L. ZUKSCHWERDT. Volume I, Part II. 10½ × 7 in. Pp. 185–329, with 43 illustrations. 1960. Stuttgart: Georg Thieme. D.M.27.

The Biochemical Response to Injury. A symposium organized by the Council for International Organizations of Medical Sciences. Edited by H. B. STONER. 8¾ × 5½ in. Pp. 467 + xii, illustrated. 1960. Oxford: Blackwell Scientific Publications. 57s. 6d.

Surgery of Theodoric. Volume II, Books III and IV. Translated by ELDRIDGE CAMPBELL, M.D., and JAMES COLTON, M.A. 8½ × 5½ in. Pp. 233 + xx. 1960. New York: Appleton-Century-Crofts Inc. $5.50.

Spinal Joint Changes and Dental Infections. By BO. E. INGELMARK, VILH. MOLLER-CHRISTENSEN, and ORE BRUNCH. Supplement 36–1 ad Vol. 38 (1959). *Acta Anatomica.* 9¾ × 7 in. Pp. 60, with 27 illustrations. 1959. Basel/New York: S. Karger. S.Fr.9.

Prophylaxie et Traitement des Escarres de Decubitus. By R. VILAIN. 9 × 7 in. Pp. 120, with 60 illustrations. 1960. Paris: L'Expansion Scientifique Française. N.F.13.50.

The Twelfth Annual Report of the Orient Hospital. 1959. Beirut: Lebanon.

Medullary Sponge Kidney. By TORE EKSTRÖM, BENGT ENGFELDT, CURT LAGERGREN, and NILS LINDVALL. 9½ × 6½ in. Pp. 73, with 28 pages of illustrations. 1960. Stockholm: Almquist and Wiksell. Sw.Kr.18.

Surgical Nursing and After-treatment. By T. EDWARD WILSON, Honorary Surgeon, St. George Hospital, Sydney. Eleventh edition. 8½ × 5½ in. Pp. 618 + x, with 361 illustrations. 1960. London: J. & A. Churchill Ltd. 30s.

The Cell of Schwann. By GILBERT CAUSEY, M.B., F.R.C.S., Professor of Anatomy, Royal College of Surgeons of England. 8½ × 5½ in. Pp. 120 + xii, with 40 illustrations. 1960. Edinburgh and London: E. & S. Livingstone Ltd. 21s.

Tetanus. Prophylaxe und Therapie. By LEO ECKMANN. 8½ × 5¾ in. Pp. 128, with 14 illustrations. 1960. Basel: Benno Schwabe & Co. Geb.Fr.16.

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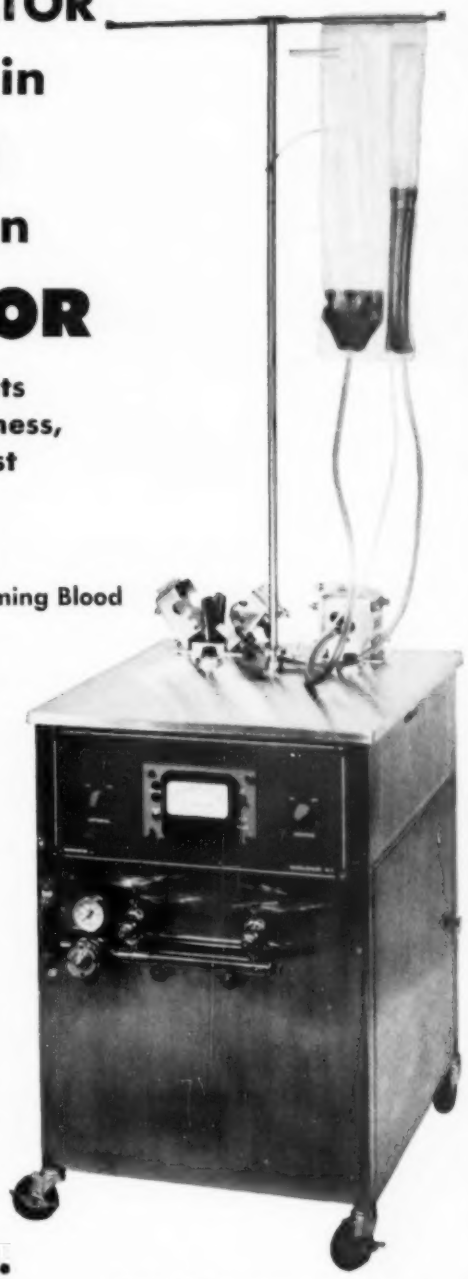

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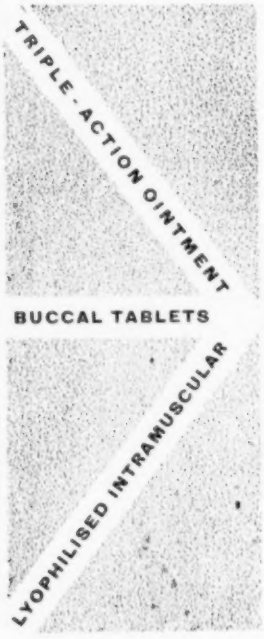

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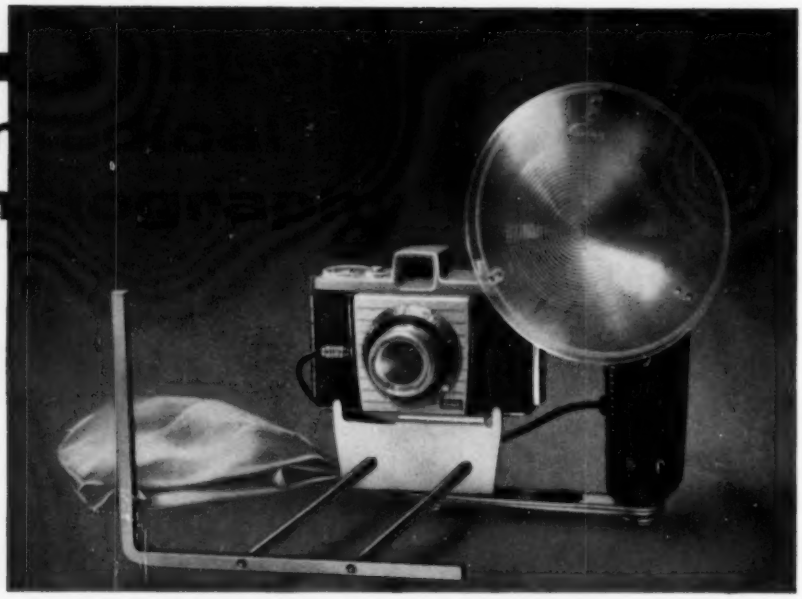

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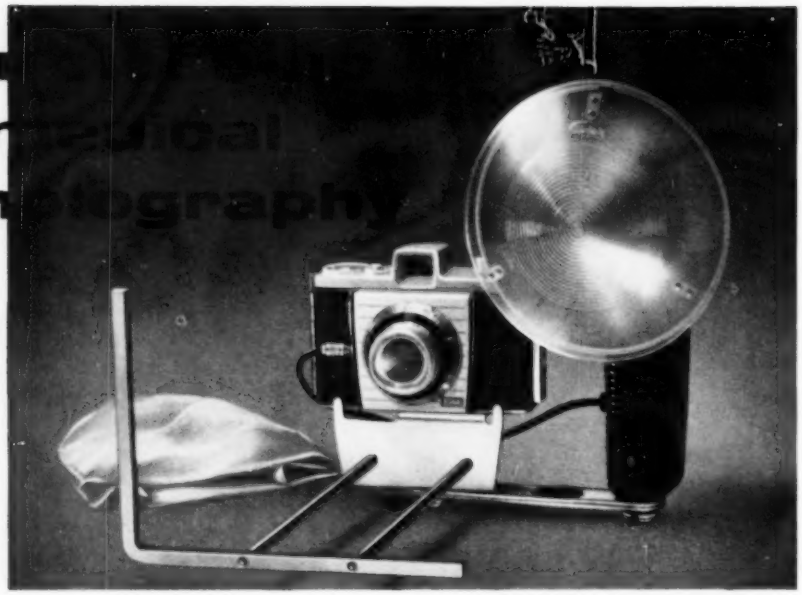

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